

A Public Prosecutor v Kenneth Fook Mun Lee (No 2)

HIGH COURT (KUALA LUMPUR) — CRIMINAL TRIAL NO 45–52 OF 2000
 AUGUSTINE PAUL J
 30 JUNE 2003

B *Criminal Law* — *Penal Code (Malaysia)* — s 300 — *Defence of automatism* — *Ingredients of* — *Whether defence of automatism may be sustained* — *Penal Code s 300*

Criminal Law — *Penal Code (Malaysia)* — s 300 — *Defence of intoxication* — *Whether evidence adduced supported such defence* — *Whether necessary to first establish that intention had been established* — *Whether such intention had been rebutted by intoxication* — *Penal Code ss 86, 300*

C *Criminal Law* — *Penal Code (Malaysia)* — s 300 — *Mens rea* — *Whether mens rea rebutted* — *Whether facts supported reduced offence* — *Penal Code ss 300, 304 (b)*

D The accused was charged with the murder of one Lee Good Yew (‘the deceased’) under s 302 of the Penal Code. According to the facts, the accused had walked towards the car of the deceased. He had knocked at the windscreen of window of the car and asked the deceased to come out. The deceased had refused and the accused then shot at her. The defence was one of automatism. In the alternative, the defence of intoxication was pleaded.

E **Held**, convicting the accused and sentencing him to eight years’ imprisonment from the date of his arrest:

F (1) Automatism refers to a state of defective consciousness in which a person performs unwilling acts. It is related to the requirement of voluntariness and mens rea. It means an act which is done by the muscles without any control by the mind, such as a spasm, a reflex action or a convulsion; or an act done by a person who is not conscious of what he is doing, such as an act done whilst suffering from concussion or whilst sleep-walking (see pp 682E–F, H–683A).

G (2) There are in law two types of automatism: (a) insane; and (b) non-insane. The basis of the distinction lies in whether the cause is internal or external to the accused. Insane automatism arises where the primary cause of the abnormality is internal to the accused and prone to recur. This condition will be classified as a disease of the mind and this is reflected in s 84 of the Penal Code. Non-insane automatism occurs where the abnormality is caused by a factor external to the accused like for example, a blow to the head, medication, alcohol or drugs. The distinction affects only the burden of proof and not the matters that require to be proved or disproved (see p 687D–F).

H (3) The alleged cause of automatism in this case was hypoglycaemia. It was not caused by a factor external to the accused. It would occur when there was a drop in the blood sugar of the accused and

- could recur. The accused's condition therefore fell under the classification of insane automatism and the burden was on him to establish the defence on a balance of probabilities (see pp 687H–688A). **A**
- (4) The facts of the case revealed that the accused fired two shots in the air at the deceased when she refused to come out of her car. He fired at the deceased as she had disobeyed his instructions. If she had obeyed him he would not have fired the shot. He pointed the gun at the crowd only when they approached him. It was the conduct of the deceased that triggered the accused to shoot at her. He only fired at the deceased when his instructions were not obeyed. It was therefore not a random shot. The sequence of events indicated that he had sufficient mental capacity to be responsive enough to make a decision. The accused was therefore sufficiently conscious of what he was doing when he fired the fatal shot as a result of a decision made by him to do so following a refusal to obey his instructions. This meant that not all the deliberative functions of his mind were absent. The defence of automatism raised by the accused could not therefore be sustained (see pp 689G–I, 691A–C). **B**
- (5) In cases where intoxication is in issue, the court must determine whether the accused had the requisite specific intent in the light of his intoxication. The court must first make a finding on the relevant mens rea before embarking on a consideration of the defence of intoxication (see p 700E–F). **C**
- (6) The onus of proof where intoxication is raised as a defence is on the accused according to s 105 of the Evidence Act 1950. The burden on the accused may be discharged by evidence of witnesses for the prosecution as well as witnesses for the defence. (see pp 700F, 701E). **D**
- (7) Intention may be inferred from the nature of the act of a person. The nature of the weapon used and part of the body to which the injury is caused may also facilitate the drawing of such inference (see p 702D–E). **E**
- (8) In this case, the accused fired the shot from outside the car into its driver's side through the front windscreen. The shot hit the upper part of the body of the driver where the vital organs of the body were situated. The shot was virtually certain to cause death and accused must be presumed to know this. The act of the accused in firing the shot was deliberate as it was done following the refusal of the deceased to open the door of the car. The deliberate use of a dangerous weapon at another leads to the irresistible inference that it was done with the intention to cause death (see p 702E–F). **F**
- (9) Furthermore, the act of the accused in firing the shot was not one of accident or negligence. Where injury caused is not the result of accident or negligence, a strong presumption arises that the injury **G**
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- A** caused was intended to be caused though this presumption may be rebutted. The circumstances in which the shot was fired therefore go beyond mere knowledge and it must be inferred or presumed that it was done so intentionally (see pp 702F–G, I–703A).
- B** (10) The presumption of the absence of intoxication which facilitated a finding that the accused acted with intention was displaced by the evidence of intoxication on record to show that he did not have the requisite intention. This meant that the act of the accused which gave rise to an inference of intention had been rebutted (see p 703D–E).
- C** (11) The facts were however sufficient to make out a case of culpable homicide not amounting to murder under s 304(b) of the Penal Code which did not require an intention to cause death or to cause such bodily injury as was likely to cause death. The accused was therefore guilty of an offence of culpable homicide not amounting to murder under s 304(b) of the Penal Code (see p 703E–F).
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[Bahasa Malaysia summary]

- E** Tertuduh telah dituduh membunuh Lee Good Yew ('si mati') di bawah s 302 Kanun Keseksaan. Menurut fakta-fakta, tertuduh telah berjalan ke arah kereta si mati. Beliau telah mengetuk cermin depan tingkap kereta dan meminta si mati keluar dari kereta. Si mati enggan berbuat demikian dan tertuduh kemudian telah menembak beliau. Pembelaannya adalah automatism. Secara alternatif, pembelaan beliau berada dalam keadaan mabuk telah diipulkan.

F Diputuskan:

- G** (1) Automatism merujuk kepada satu keadaan kesedaran yang defektif di mana seseorang melakukan tindakan yang tidak diniatkan. Ia berkaitan keperluan kesukarelaan dan mens rea. Ia bermaksud satu tindakan yang dibuat oleh otot-otot tanpa kawalan minda, seperti kekejangan otot, tindakan refleks atau konvulsi; atau satu tindakan yang dibuat oleh seseorang yang tidak sedar apa yang beliau lakukan, seperti satu tindakan yang dilakukan bila mengalami konkusi atau semasa berjalan dalam tidur (lihat ms 682E–F, H–683A).
- H** (2) Terdapat dua jenis automatism dalam undang-undang: (a) hilang akal; dan (b) tidak hilang akal. Asas perbezaannya terletak di mana sama ada sebabnya adala dalaman atau luaran kepada tertuduh. Automatism hilang akal timbul di mana sebab utama keadaan tidak normal tersebut adalah dalaman kepada tertuduh dan boleh berulang. Keadaan ini akan diklasifikasikan sebagai satu penyakit minda dan ini dinyatakan dalam s 84 Kanun Keseksaan. Automatism tidak hilang akal berlaku bila keadaan tidak normal disebabkan oleh satu faktor luaran kepada tertuduh
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seperti contoh, satu pukulan dikepala, pengambilan ubat, alkohol atau dadah. Perbezaan tersebut hanya melibatkan beban pembuktian dan bukan perkara-perkara yang perlu dibuktikan atau tidak (lihat ms 687D–F).

(3) Sebab automatism yang dikatakan dalam kes ini adalah hypoglycaemia. Ia tidak disebabkan oleh satu faktor luaran tertuduh. Ia akan berlaku apabila paras gula tertuduh menjadi rendah dan boleh berulang. Keadaan tertuduh oleh itu terangkum di bawah kelas automatism hilang akal dan beban terletak atas beliau untuk membuktikan pembelaan atas imbalan kebarangkalian (lihat ms 687H–688A).

(4) Fakta-fakta kes menunjukkan bahawa tertuduh telah menembak dua das ke udara kepada si mati apabila si mati enggan keluar dari kereta beliau. Beliau telah menembak si mati kerana tidak mematuhi arahannya. Jika si mati mematahinya beliau tidak mungkin telah menembak si mati. Beliau mengacukan pistol ke arah orang ramai hanya apabila mereka mendekati beliau. Ia adalah perbuatan si mati yang menyebabkan tertuduh menembaknya. Beliau hanya menembak si mati bila arahan beliau tidak dipatuhi. Oleh itu ia bukan satu tembakan rambang. Urutan kejadian menunjukkan bahawa beliau mempunyai kapasiti pemikiran yang memadai untuk bertindak responsif dalam membuat satu keputusan. Tertuduh oleh itu cukup sedar apa yang beliau lakukan apabila beliau melepaskan tembakan maut tersebut akibat daripada keputusan yang dibuat oleh beliau untuk melakukan sedemikian berikutan keengganan untuk mematuhi arahan beliau. Ini bermakna bukan semua fungsi perundingan minda beliau tiada. Pembelaan automatism yang ditimbulkan oleh tertuduh tidak boleh dikekalkan (lihat ms 689G–I, 691A–C).

(5) Dalam kes-kes di mana keadaan mabuk menjadi persoalannya, mahkamah perlu menentukan sama ada tertuduh mempunyai niat spesifik yang diperlukan berkaitan keadaan mabuk beliau. Mahkamah pertama sekali perlu membuat penemuan atas mens rea yang relevan sebelum membuat satu pertimbangan pembelaan berkeadaan mabuk (lihat ms 700E–F).

(6) Beban pembuktian di mana keadaan mabuk ditimbulkan sebagai satu pembelaan adalah ke atas tertuduh menurut s 105 Akta Keterangan 1950. Beban atas tertuduh boleh dilepaskan dengan keterangan saksi-saksi bagi pihak pendakwa dan juga saksi-saksi bagi pihak pembela (lihat ms 700F, 701E).

(7) Niat boleh diandaikan daripada sifat perbuatan seseorang itu. Sifat senjata yang digunakan dan bahagian badan yang mana berlakunya kecederaan boleh juga mengenalpastikan penemuan inferens tersebut (lihat ms 702D–E).

(8) Dalam kes ini, tertuduh telah menembak dari luar kereta ke sebelah bahagian pemandu melalui cermin depan. Tembakan

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- A** tersebut terkena pemandu di mana terletak organ-organ penting badan. Tembakan tersebut sudah tentu menyebabkan kematian dan tertuduh mestilah dianggap mengetahuinya. Perbuatan tertuduh menembak adalah sengaja kerana ia dilakukan setelah si mati enggan membuka pintu kereta. Penggunaan senjata merbahaya yang disengajakan ke atas yang lain membawa kepada inferens yang tidak dapat dibendung bahawa ia telah dilakukan dengan niat untuk menyebabkan kematian (lihat ms 702E–F).
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- C** (9) Tambahan pula, tindakan tertuduh menembak bukan satu kemalangan atau kecuaiian. Di mana kecederaan berlaku bukan akibat daripada kemalangan atau kecuaiian, satu andaian yang kukuh timbul di mana kecederaan yang berlaku memang diniatkan walaupun andaian ini boleh dipatahkan. Keadaan-keadaan di mana tembakan dibuat oleh itu melampaui pengetahuan sahaja dan ia mestilah diandaikan atau dianggap bahawa ia telah dilakukan dengan sengaja (lihat ms 702F–G, I–703A).
- D** (10)Tanggapan ketiadaan keadaan mabuk yang membantu penemuan bahawa tertuduh bertindak dengan niat telah digantikan dengan keterangan berkeadaan mabuk atas rekod untuk menunjukkan bahawa beliau tidak mempunyai niat yang diperlukan. Ini bermakna bahawa tindakan tertuduh yang menimbulkan satu inferens terdapat niat telah dipatahkan (lihat ms 703D–E).
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- F** (11)Fakta-fakta bagaimanapun adalah mencukupi untuk membentuk satu kes membunuh tanpa niat di bawah s 304(b) Kanun Keseksaan yang tidak memerlukan satu niat untuk menyebabkan kematian atau untuk menyebabkan kecederaan kepada badan yang berkemungkinan menyebabkan kematian. Tertuduh oleh itu adalah bersalah kerana satu kesalahan membunuh tanpa niat di bawah s 304(b) Kanun Keseksaan (lihat ms 703E–F).]

Notes

- G** For cases on the Penal Code (Malaysia) s 300, see 4 *Mallal's Digest* (4th Ed, 2000 Reissue) paras 1155–1168.

Cases referred to

- H** *Abdullah bin Jacomah v PP* [2002] 6 MLJ 324 (refd)
Aladin v PP [1964] 1 MLJ 345 (refd)
Basdev v State of Pepsu AIR 1956 SC 488 (folld)
Behari v State AIR 1953 All 203 (refd)
Bhagat Singh v Emperor 121 IC Lah 726 (refd)
Bratty v Attorney-General for Northern Ireland [1963] AC 386 (folld)
- I** *Broome v Perkins* (1981) 85 Cr AR 321 (refd)
Chan Chor Shuh v PP [2003] 2 MLJ 26 (refd)
Dil Mohammad v State (1941) 21 Pat 250 (refd)

- Director of Public Prosecutions v Majewski* [1977] AC 443 (refd) **A**
Emperor v Mt Dhirajia AIR 1940 All 486 (refd)
Francis a/l Anthonysamy v PP [2003] 2 MLJ 49 (refd)
Gyarsibai v State AIR 1953 MB 61 (refd)
Hasta Ismail v Emperor AIR 1937 Lah 593 (refd)
Haynen v Ministry of Transport (1988) 3 CRNZ 587 (refd)
Inder Singh v Crown (1928) ILR 10 Lah (refd) **B**
Indra Wijaya Ibrahim v PP [1995] 2 SLR 42 (refd)
Janardan Prasad v State of Rajasthan 1977 Cr LJ 68 (refd)
Juma'at bin Samad v PP [1993] 3 SLR 338 (folld)
Kali Charan Mukharji, In re 11 Cal LR 232 (refd)
Kanji v State (1953) Cr LJ 434 (refd) **C**
Kennedy v HM Advocate (1944) SC (J) 171 (refd)
Liew Koh Tai v PP [1965] MLJ 54 (refd)
MA Clyde v Wong Ah Mei & Anor [1970] 2 MLJ 183 (refd)
Munah binti Ali v PP [1958] MLJ 159 (refd)
Munusamy v PP [1987] 1 MLJ 492 (refd)
Nga Tun Baw v Emperor (1912) XVII IC 800 (refd) **D**
PP v Awang Raduan bin Awang Bol [1998] 5 MLJ 460 (refd)
PP v Kenneth Fook Mun Lee (No 1) [2002] 2 MLJ 563 (refd)
PP v Mohamed Noor Jantan [1979] 2 MLJ 80 (refd)
PP v Ramasamy a/l Sebastian [1991] 1 MLJ 75 (refd)
PP v Tan Ho Teck [1988] 3 MLJ 264 (refd)
PP v Teo Heng Chye [1989] 3 MLJ 205 (refd) **E**
Pal Singh v State AIR 1917 Lah 226 (refd)
Police v Bannin [1991] 2 NZLR 237 (refd)
Purshoftam Ishvar Amin v Emperor AIR 1921 Bom 3 (refd)
R v Barry Douglas Burgess (1991) 93 Cr App R 41 (refd)
R v Burr [1969] NZLR 736 (refd) **F**
R v Cottle [1958] NZLR 999 (refd)
R v Doherty 16 Cox CC 306 (refd)
R v Hennessy [1989] 2 All ER 9 (refd)
R v Monkhouse (1849) 4 Cox CC 55 (refd)
R v Nedrick [1986] 3 All ER 1 (refd)
R v Quick [1973] QB 910 (refd) **G**
R v Sheehan [1975] 2 All ER 960 (refd)
R v Stone (1999) Can Sup Ct Lexis 30 (refd)
R v Upendra 19 CWN 653 (refd)
Rama v State AIR 1969 Goa 116 (refd)
Ranji v State 1952 RLW 145 (refd) **H**
Rajeevan Edakalavan v PP [1998] 1 SLR 815 (refd)
Ramamoorthy v PP [1948] MLJ 43 (refd)
Ryan v R (1967) 121 CLR 205 (refd)
S Nagarajan s/o Kuppusamy v PP [1999] 2 SLR 111 (refd)
Sim Eng Teck v PP [1998] 3 SLR 818 (refd)
State of Madhya Pradesh v Ram Prasad AIR 1968 SC 881 (refd) **I**
Suba Singh v PP [1962] MLJ 122 (refd)
Suradet v PP [1993] 3 SLR 265 (refd)

- A *Tan Hung Song v Rex* [1951] MLJ 181 (refd)
Tham Kai Yaw & Ors v PP [1977] 1 MLJ 174 (refd)
Wong Chooi v PP [1967] 2 MLJ 180 (refd)

Legislation referred to

- B Criminal Procedure Code ss 347, 348
 Evidence Act 1950 ss 31, 105
 Penal Code ss 39, 84, 85(2)(a), (b), 86(2), 299, 300 illustration (d), 302, 304(b)

- C **Appeal from:** Arrest Case No B81–24 of 2000 (Magistrate’s Court, Kuala Lumpur)

Abdul Karim bin Abdul Jalil (Abdul Wahab bin Mohamad, Ahmad Fairuz bin Zainol Abidin and Rosidah bte Abu Bakar with him) (Attorney General’s Chamber’s) for the prosecution.

- D *Muhammad Shafee Abdullah (K Kumaraendran, Wong Kian Kheong, Leena Ghosh, Rabinder Singh and Dev Kumaraendran with him)* (*Shafee & Co*) for the accused.

Augustine Paul J: Kenneth Fook Mun Lee @ Omar Iskandar Lee bin Abdullah (‘the accused’) was charged with murder under s 302 of the Penal Code. The charge reads as follows:

- E Bahawa kamu pada 22 Ogos 2000 jam lebih kurang 7.55 malam di Jalan Istana Baru, di dalam Daerah Brickfields, di dalam Wilayah Persekutuan, Kuala Lumpur telah melakukan bunuh dengan menyebabkan kematian Lee Good Yew (Kad Pengenal No 480520-07-5432) dan dengan itu kamu telah melakukan suatu kesalahan yang boleh dihukum di bawah s 302 Kanun Keseksaan.

He claimed trial to the charge.

- G The events that led to the unfortunate loss of a life on 22 August 2000 began with a lunch the accused had at the Weng Seng Restaurant at No 93, Jalan 8, Batu 9, Cheras owned by Ng Kiew Meng (‘PW14’). The accused came alone in his Volvo car to the restaurant at about 1.45pm and joined three of his friends who were already there. PW14 knows the name of one of the three persons as Tan Ah Chai (‘DW5’). They ordered lunch consisting of fish, fried eggs, vegetable and rice. The accused and two of his friends consumed beer. They drank a total of ten large bottles of Tiger beer of which the accused took five to six glasses. They were in the restaurant till 7pm, having ordered the last beer at about 6pm, PW14 said that till the accused left in his car, he appeared normal and did not look drunk. PW14 said in cross-examination:

- I Saya ada lihat Kenneth Lee balik ke keretanya. Semasa di restoran, Kenneth Lee ada pergi ke tandas. Lebih kurang tiga kali. Dia jalan normal sahaja. Saya tidak nampak dia mabuk pada masa itu. Sebelum Kenneth Lee balik, dia hanya kata, bye-bye kepada saya. Dia nampak normal. ... Kenneth Lee tidak marah dan tidak misbehave.

With regard to the journey back to Kuala Lumpur from his restaurant, PW14 said: **A**

Jalan ke restoran saya jalan biasa. Jalan itu seperti jalan di kampung. Cheras village starts from this area. It is the oldest part of Cheras. Untuk pergi ke Kuala Lumpur dari restoran perlu melalui banyak lorong, pusing ke kanan kiri. Saya sendiri pandu kereta. Saya pergi ke Kuala Lumpur kerap kali. Saya biasa pergi ke Pudu. Saya perlu melalui dua tol. **B**

Rahim bin Harun ('PW4'), a part-time taxi driver, Lim Chan Chien (PW3), a tax supervisor and Abdul Rahim bin Senin (PW6), a photographer with the *Star Papyrus Publications* gave evidence on what they saw at Jalan Istana Baru just before 8pm on the same day. When PW4 drove his taxi into Jalan Istana Baru he saw a red Volvo car parked across the road. He saw the accused standing in the middle of the road. He stopped a motorcyclist. However, the motorcyclist sped off immediately. The accused then bent down and took out a gun from his ankle holster and fired two shots in the air. At that time, PW4 was about 20ft away from the accused. The accused then stopped PW4 by pointing the gun. PW4 stopped his taxi immediately. The accused then came towards the taxi. The window screen of the taxi was down. The accused came towards the door of the taxi and pointing the gun towards PW4's head told him in a loud and fierce voice, 'Get out'. PW4 got out. The accused told him, 'You stay here'. The accused then pointed the gun in the direction of PW4's head and stomach. He was holding the gun in his right hand. The accused told PW4 to stand straight. When PW4 did so, the accused signalled to him the 'Good' sign by showing the thumb. The distance between the accused and PW4 was about two feet. PW4 stood straight for about ten minutes. PW4 did not get any smell from the accused. The traffic at that time was not heavy. The accused then looked towards the right of PW4 and saw a car moving slowly. He then walked towards the car, a light green Perdana, which was about ten feet away from PW4's taxi. When the accused approached the Perdana, he knocked at the window of the car. PW4 took this opportunity and drove off. He said that the accused looked normal and was steady and not staggering when walking. PW3, who was driving back home after 7.30pm on that day, heard the sound of a gunshot at the junction of Jalan Dewan Bahasa and Jalan Istana Baru. She saw a Volvo parked across the road in the centre of Jalan Istana Baru obstructing cars from going through the road. She saw a taxi to the left of the Volvo. The driver of the taxi had come out. The accused was standing beside the taxi. He was holding a gun. As PW3 was following some other cars ahead of her, she saw the accused moving towards the cars. He fired two shots. A Perdana car was on her left. PW3 then said: **C**
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I saw the man with the gun shooting at the driver of the Perdana. Prior to shooting, the man with the gun knocked at the windscreen driver side of the window of the car with the gun and asked the driver to come out. I cannot recall how many times he knocked. But it was more than once. The driver of the car did not come out. I did not see the driver. But I saw somebody inside the car. I did not know what happened inside the car. After the man knocked at the car, he walked to the front right side of the car and just fired a shot. The Perdana then moved forward and hit the headlight of the Volvo. ... I know **I**

- A** that the man wanted the driver of the Perdana car to come out because he tried to open the door. This was more than once. I did not hear any spoken words.

In her cross-examination, PW3 said:

- B** He was trying to open the door of the car. I could not hear the voice of the accused. He looked angry. ... I say that the accused was trying to get the driver of the Perdana out as he was trying to open the door. He knocked at the screen and the side window with his gun. He then moved in front of the Perdana. He stood there for a while and shot. I can't say for how long he stood. I can't recall which hand he used to pull the trigger. I still cannot recollect which hand he used. He put his hand forward with the gun and shot. I can't recall whether he aimed and shot.

- C** PW3 then followed another car and drove off. PW6 was riding his motorcycle at about 7.55pm. At Jalan Istana Baru, he saw a Volvo parked across the road and the accused holding up a few cars as if in a roadblock. PW6 saw a taxi and another car going through. The accused then went towards the direction of the Perdana. PW6 then said:

- D** Orang Cina seperti pegawai polis menahan kereta semasa roadblock. Lelaki Cina itu menuju ke satu kereta Perdana. Dia seperti macam sedang tahan kereta dan bercakap sesuatu yang saya tidak tahu. Dia cakap dengan orang dalam kereta Perdana. Saya tak nampak siapa yang dalam kereta. Dia mengacu-acukan pistol kepada kereta Perdana. Saya nampak dia mengacu pistol. Jarak antara saya dan Perdana lebih kurang 15m. Cahaya di kawasan itu sangat terang. Kereta-kereta yang berhenti memasang lampu. Ada lampu jalan. Dia melepaskan tembakan ke arah cermin depan kereta Perdana. Pada masa itu, lelaki Cina itu berada di tepi kereta Perdana di tayar depan. Lelaki Cina itu berdiri di tepi tayar di kanan. Selepas saya nampak lelaki Cina tembak, saya nampak kereta Perdana itu berjalan perlahan-lahan dan berlanggar kereta Volvo di bahagian belakang.

PW6 then added:

- E** Saya menjerit kuat. Saya menjerit kepada OKT. Kemudian OKT melepaskan tembakan ke arah saya. Saya nampak OKT mengarahkan pistol ke arah saya. Dia melepaskan satu das tembakan. Tetapi tidak terkena kepada saya. Saya menjerit kepada orang-orang yang berada di depan dan belakang kereta supaya keluar daripada kereta masing-masing dan melarikan diri. Mereka meninggalkan kereta masing-masing. Keadaan lalu lintas pada ketika itu sesak. Orang keluar dari kereta dan lari. Saya pun melarikan diri. Saya lari ke belakang ke arah Jalan Dewan Bahasa. OKT melepaskan satu lagi tembakan. Saya kurang pasti dia tembak ke arah mana. Ada lagi satu tembakan.
- F** Tembakan ini ke arah trak askar. Ada dua hingga tiga tembakan yang dilepaskan ke arah trak askar. Pada masa itu, trak askar berada di tengah jalan di bawah jalan flyover. Ini di depan Bangunan MAA — tanda MAA di ID5 (saksi dirujuk kepada P44 (17), (18) dan (20)). Inilah trak askar yang saya maksudkan. Selepas menembak ke arah trak askar, OKT menuju ke arah trak askar. OKT menuju ke arah Bangunan MAA. Tidak terdapat banyak kereta di atas jalan di depan Bangunan MAA kerana terdapat anggota polis yang sedang mengawal lalu lintas di situ. Ada kereta yang berhenti di jalan menuju ke atas flyover. Saya nampak OKT turun ke Bangunan MAA. OKT melalui jalan tengah. Selepas itu, OKT berlegar-legar di depan Bangunan MAA.

OKT berlegar-legar di atas kawasan parking di Bangunan MAA. Ada ramai orang di tepi jalan. OKT pergi balik ke jalanraya. Selepas itu, OKT menuju ke sebuah teksi. Dia berdiri seperti sedang menahan teksi itu. Teksi itu berhenti. OKT mencari drebar teksi. Teksi itu tidak ada pemandu pada ketika itu. Selepas itu, OKT bersandar di teksi itu. Semasa OKT bersandar di teksi, beliau tidak buat apa-apa. Selepas itu pegawai polis pun sampai. Pegawai polis kemudian tangkap OKT. Apabila OKT bersandar di teksi, pistol masih di tangannya. OKT cuba tembak tetapi tidak ada peluru. Boleh dengar. Orang ramai yang lihat kejadian menjerit dan berkata 'Peluru dah habis'. Saya kurang pasti OKT cuba tembak ke arah mereka. Sebelum OKT cuba menembak dan tidak ada peluru, orang ramai cuba tangkap dia. Selepas itu, anggota polis tangkap OKT. Selepas polis berjaya tangkap OKT, orang ramai telah memukul OKT. Pihak polis menahan orang ramai. Saya ada di situ.

A

B

C

While under cross-examination, PW6 agreed that the behaviour of the accused showed that he could not see clearly. He also said:

I agree that the behaviour of the accused gave me the impression that he was confused.

D

PW6 said that after shooting at the Perdana car, the accused while walking towards the MAA Building was:

... .. walking aimlessly as if he was lost.

PW6 then continued:

E

While the accused was walking down the road after the Perdana incident, he was seated at the side of the road for a while. When he got up from his seated position, I threw something at him. He was seated for a short while. For about 30 seconds. Maybe less. I threw a stone at him. Others also threw things at him. He then started walking aimlessly. He did not react to the stone thrown at him. But the stone did not hit him. He appeared to be unconscious of the surroundings.

F

At the time of the arrest, the police pounced on him and handcuffed him. At the time of the arrest, the accused was not in a fit-like shivering and shaking his body. I saw his behaviour. ... I do not agree that at the time of the arrest, the accused was shivering.

G

On the same day at about 8.05pm, Inspector T Sivakumar ('PW7') was informed of the incident by Lance Corporal Izzuddin bin Hamidon ('PW11'). Both of them proceeded to the scene on a motorcycle and arrived there at about 8.10pm. They saw the accused standing behind a taxi and next to an army truck. He was holding a gun. As PW7 said:

H

Dia memegang pistol dan sedang mengacukan ke arah orang ramai. Orang ramai berada di sebalik pokok dan belukar di jalan itu.

Setelah saya pastikan lelaki itu memiliki pistol, saya pergi lebih dekat. Lelaki itu berjalan menghampiri kereta tentera di sebelah. Kemudian lelaki itu telah mengacukan semula ke arah orang ramai. ...

I

Punca lelaki tersebut mengacukan pistol ke arah orang ramai kerana orang ramai cuba menghampiri lelaki tersebut. Apabila lelaki itu mengacukan, mereka akan lari di belakang.

A In this regard, PW7 said in his cross-examination:

Apabila OKT mengacu, orang ramai pergi belakang. Ini berlaku sekali, dua kali. Rasa-rasanya lebih dari sekali. Saya ada nampak seorang lelaki berlari melintas jalan dan kemudian melintas semula pada jarak lebih kurang 40m dari tempat lelaki yang memegang pistol itu. Seorang sahaja yang buat itu. Ini bermaksud mereka cuba menangkap lelaki itu. Apa yang terlintas di hadapan lelaki itu dan yang menarik perhatian lelaki itu, dia akan mengacu ke arah tersebut. ... OKT pegang pistol dengan tangan kanan.

B

PW11 took possession of the gun ('exh P9'). PW7 arrested the accused. PW7 then took away the accused in a patrol car. They arrived at the police station at 8.20pm. There, PW7 found a holster ('exh P14') on the left leg of the accused. The magazine ('exh P9A') of exh P9 was found to be empty.

C In his cross-examination, PW7 said:

From my observation, the accused was steady before his arrest. Saya sampai dahulu di pejabat. Sebelum IO datang, OKT dijagai oleh anggota polis. OKT ditempatkan di bilik gerakan. OKT berbaring dalam bilik. Lebih kurang 10–15 minit selepas itu, OKT telah membuang air besar kerana bau. Bila ini berlaku saya tidak tahu. Tetapi saya sedar berkenaan ini semasa OKT sudah pun terbaring di dalam pejabat. OKT ada membuang air besar dan air kecil. OKT terbaring tidur dan berdengkur lebih kurang 20 minit. Ada besar kemungkinan OKT membuang air besar dan air kecil pada masa itu. Saya tidak lihat OKT secara continuous. Saya tidak perhatikan OKT menggigil. Saya tak ingat sama ada seorang bernama Ramayah a/l Muniandy (atau pun Niko) ada dalam pertahanan pada masa itu. Saya tidak tahu sama ada Niko adalah orang yang bersihkan OKT. ...

D

E

Saya tahu berkenaan OKT terbang air besar dan air kecil bukan cara siasat tetapi dengan cara terbau. Tidak mungkin OKT membuang air besar dan air kecil sebelum itu. Semasa OKT dibawa masuk ke Balai Polis, saya tidak nampak sebarang makanan atau minuman diberi kepadanya.

F

In the meanwhile, Lance Corporal Razali bin Kadri ('PW17') and Lance Corporal Hassan bin Bujang had gone, upon receipt of information after 7.45pm, to the scene at 7.55pm, PW17 saw a Perdana car there. The front windscreen had a suspected bullet mark. He found the body of a female person inside the car. She was unconscious and was suspected to have been shot. He saw blood marks on her chest. With the help of some members of the public, he removed the body. He could see the face of the female clearly. He said that the body was that of a Chinese person. He identified the person as the person in two photographs ('exh P4 (31) and (32)') shown to him which had earlier been identified by Lee Yoot Fong ('PW2') as the body of her sister, Lee Good Yew ('the deceased'). Members of the public then sent the body to the hospital.

G

H

Prof Dr K Nadeson ('PW16'), a forensic pathologist attached to the University Hospital, Kuala Lumpur, carried out an autopsy on the deceased on 23 August 2000 at 1.30pm. His external examination of the deceased showed that her frock had a tear 3cm by 1.5cm on the right mid-front of the chest area and the right cup of the brassiere showed a roughly oval shaped tear measuring 2cm by 1cm. The frock was stained with blood around the area of the tear. The internal examination showed a lacerated wound 3.7cm

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by 2cm on the mid-front of the right chest. The wound was placed obliquely horizontal situated 23cm below the tip of the right shoulder and about 2cm from the mid-chest. In the words of PW16:

A

The outer end was at a straightly high level than the inner end and there was an obvious marginal aberration towards its outer end. There was a track originating from the inner end of this wound. It had penetrated into the chest cavity to the right sixth inter costal place. Also fracturing the right free border of the sternum. Then through and through the right ventricle of the heart. Then through the hilum of the left lung and through and through the thoracic aorta and then entered the left lumbar area fracturing and gutter in the left side of the 11th thoracic vertebra.

B

PW16 said that a ripped off jacket of a rifled weapon bullet was found lodged in the fractured 11th thoracic vertebra and a severely deformed lead core of the bullet was found lodged in the left lumbar area of the back closer to the first lumbar vertebra underneath the skin of the deceased ('exhs 54 and 55'). With regard to the cause of death, he said:

C

All the injuries were necessarily fatal in nature. The injury to the heart, lung and aorta gave no chance of survival. They were all caused by a single bullet traversing through these areas.

D

The cause of death is due to bullet wound to the chest with injuries to vital organs.

.....

The injuries sustained by the deceased were necessarily fatal. Nothing could have saved her.

E

PW16 handed over exhs P54, P55 and some other items to ASP Wan Kok Chai ('PW15'), the officer who led the investigation in this case.

PW15, upon being instructed to investigate into this case, went to the police station at 10.05pm. PW7 informed him about the arrest of the accused. PW15 saw the accused sleeping on the floor. He then left for the scene of the crime at Jalan Istana Baru and arrived there at about 10.20pm. PW15 then said:

F

At the scene, I found two cars parked on the right hand side of the road. One was a Proton Perdana BFW 6366 parked facing Istana Negara. The other was a red Volvo WS 9 facing the opposite direction that is against the flow of traffic. I examined both the cars. I found there was a bullet hole on the front windscreen on the driver's side of the Proton Perdana. I also found the mirror of window on the right hand side was broken. I then instructed the photographer L/Kpl Kumaguru (SP5) to take photographs. As for the Volvo WS 9, I found that there was no damage to the car. The left hand front bumper of Perdana was dented. ... About 470m away at Dewan Bahasa, I found an army landrover ZB 4970 parked by the side of the road. On examining the landrover, I found there was a bullet hole on the left back window.

G**H**

On the instructions of PW15, Chief Inspector Nor Azman bin Hj Mohd Nor ('PW9') picked up some exhibits from the scene including bullet casings and bullet heads ('exhs P24 to P42'). He also took four swabs from both the hands of the accused ('exh P42 A-D'). PW15 returned to the

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- A** police station at 11.15pm and found the accused seated on the floor and handcuffed. As PW15 said:

The moment the accused saw me, he got up. He appeared very angry and was scolding everybody who was near him.

- B** With the assistance of Sergeant Ang and Lance Corporal Kumar, PW15 took the accused to the hospital at 12.30am. The accused was referred to Dr Bhawani Veloo ('PW8'). PW15 gave PW8 a consent form for the purpose of taking the accused's blood and urine samples. The samples were taken ('exhs P19 and P20B'). With regard to the custody of the samples, PW15 said:

- C** I kept both the samples inside the refrigerator in the Serious Crimes Office. Both the samples were placed in a plastic packet and placed separately from other exhibits.

- D** PW15 attended the autopsy conducted on the deceased by PW16 who handed over some exhibits to him. PW15 packed and sealed all the exhibits that he had received. He handed them to Pua Hiang ('PW13'), a chemist attached to the Chemistry Department, for examination. PW15 received copies of the chemist report and post-mortem report. The post-mortem report ('exh P72') was served on the accused on 5 February 2001. In describing the condition of the accused when handed over to him, PW15 said:

- E** From the time I was handed the accused I did not at any time give any caution. I did not caution him at the time he was handed to me because of his condition. The accused appeared aggressive and was under the influence of alcohol. As such, I found it not proper to caution him. He had a strong smell of alcohol. The first time the accused was cautioned was when his cautioned statement was taken on 23 August 2000 by ASP Thomas Woon.

- F** In this regard, he said in cross-examination:

When I said the accused was under the influence of alcohol, he had a strong smell of alcohol. He appeared aggressive verbally. He was using vulgar words to find out why he was arrested. He did give me the impression that he did not know for what reason he was arrested. ...

- G** Throughout the night of the 22nd, the accused referred to me as Khoo Ah Si. Despite my telling him, he insisted that I was Khoo Ah Si. Khoo Ah Si is bigger in size than me. I cannot comment whether he is similar to me.

He also said:

- H** On the night of the 22nd, I did not question the accused much as he appeared aggressive and smelt of alcohol. Whether or not he could remember or recollect things is not within my knowledge. I agree that the accused maintained that he could not remember anything.

In further cross-examination, PW15 said that he could not find any witness to explain how the Volvo came to be in the position in which it was found.

- I** He then said:

There were no tyre marks on the road. There was no evidence of a violent driving.

In describing the route to be taken from Cheras to Jalan Istana Baru, PW15 said: **A**

I am familiar with the road one has to take from the restaurant in Cheras to Jalan Istana. From the restaurant, one has to come out to the main road of Jalan Cheras and turn into Connaught Highway. There is a toll at Connaught Highway where payment has to be made on the spot. After the toll, one has to proceed straight and make a turn down to the main road of Jalan Sungei Besi passing by the old airport on the left. Then one has to turn left where we can join up with Jalan Dewan Bahasa and from there join up with Jalan Istana Baru. It would involve one going into the road in P3B. From there, one would join the main road at Jalan Istana. **B**

For the Volvo to be going against the flow of traffic, it would either have come in from Jalan Istana and turn left into Jalan Istana Baru. That would mean one has to make a very sharp turn in order to go against the flow of traffic. Or one would have come down from Jalan Bellamy which is shown in P13H. Coming down from Jalan Bellamy would already be facing against the traffic. There is no other possibility. **C**

There is only one toll as I had mentioned. I agree that before the Connaught toll there is the Cheras toll. That is also a pay and go toll. There was no report of any mischief on both the tolls. There was also no report of dangerous driving by a car fitting the description of the Volvo from 7–8.00pm on the 22nd. **D**

With regard to the consent form in respect of the blood and urine samples, PW15 said: **E**

I prepared a kind of consent form. It was type-written on a piece of paper. I started it with the report number and the wording is more or less like this: ‘I, Kenneth Fook Mun Lee, hereby give my consent to have my urine and blood samples taken for examination.’ It was signed by Kenneth Lee and me. This was in my office. This is the form that I gave to the doctor and which is now not in my possession. The form was prepared before 12.30am. **F**

.....

The letter of consent that I prepared was not returned to me. I am definite of this. Had the copy been returned to me, I would have enclosed it in my investigation papers. There is nothing of that sort in my investigation diary. **G**

In his re-examination, PW15 said that he had telephoned the accused’s wife. As he said:

In the later part of the investigation, I called the accused’s wife. Her name is Noor Salasawati. I called her in her house phone. I cannot remember the number. (Witness applied to refer to his statement. Allowed.) The number I called is 03-7555671. **H**

As he said further:

When I saw the accused for the second time, he was conscious. I say so because the moment he saw me entering, he stood up and talked to me. He called me Khoo Ah Si. He made a request to make a phone call to Musa Hitam. I did not accede to that. **I**

A PW8 said that on 23 August 2000 at about 1.15am, she was asked to examine the accused by PW15. She added:

B He was brought in handcuffs. He was dressed in a pair of shorts and short sleeved shirt. Initially before the patient's file reached my desk, the doctors on call heard a commotion outside the doctor's room in the waiting area. The accused was highly agitated, restless and demanding to be freed of his handcuffs. He was also heard to be verbally abusing the other patients.

C When he was brought in to my room, he was very restless. He was demanding to contact his wife. He was demanding for no one in particular. I asked him to have a seat. That was when ASP Wan handed to me the police report which is the Police Form 59 and the request from the police for the blood and urine alcohol level to be done. When I asked the accused to sit he sat on the patient's chair. He was restless, agitated and verbally abusive.

She also said:

D Clinically, the accused was under the influence of alcohol but was lucid. It means that he was still alert at the time he was brought to the hospital.

She found the accused to have some superficial abrasions. He was conscious with stable vital signs. She then said:

E That means that his blood pressure and pulse rate were within normal range. His blood pressure was 130 by 80mm of mercury. His pulse rate was 102 per minute. His temperature was 37C. This is normal. He was not pale or jaundiced. There was a strong alcohol odour on his breath. His pupils were equal and reactive to light. That means normal. His face was flushed and his hands were unsteady. His heartbeat was normal. His lungs were clear. His abdominal examination was normal. His central nervous system examination revealed no focal deposit.

F I could see the injuries that he had sustained.

G I elicited some history from the accused. He conversed with me. He was very agitated, demanding to be freed of his handcuffs and demanding to see his wife. His speech at that time was slurred. When I interviewed him, he responded to the questions I asked. He stated his full name. I asked him what was the reason he was brought by the police. He said that he had consumed alcohol in the evening of 22 August 2000 and he was not aware of the reason why the police had arrested him. I then asked him, his past medical history. He said that he had no medical illnesses previously. I then asked him whether he had any psychiatric or mental illnesses previously which he also denied. I then asked him whether he was on any medication. He said that he was not on any medication.

H

With regard to the taking of the blood and urine samples, PW8 said:

I On the police report is a consent form whereby the accused had to sign his consent for his blood and urine to be taken. I explained the procedure to him and asked if he understood. He said he understood thereby giving me his consent to take his blood and urine sample to be tested for alcohol. I took the blood sample. The accused co-operated but he also insisted on somebody informing his wife.

The accused then took a piece of paper and wrote a note on it to one Prof Dr Ernest Yeoh and gave it to PW8. The note was tendered and marked as exh P17. It reads as follows:

TO:PROF DR
ERNEST YEOH

MY NAME IS KENNY LEE. I HAVE BEEN ARRESTED BY THE
POLICE SINCE 5.00 SOMETHING THIS EVENING.

PLEASE INFORM MY WIFE 7555671 & HAVE HER CALL MUSA
HITAM.

SGD

(KENNETH)

The blood and urine samples were then taken ('exhs P19 and P20B'). They were labelled and sealed by Yuzaini Rozmi bin Yusoff ('PW12'). PW8 concluded her examination-in-chief by explaining her reasons for saying that the accused was under the influence of alcohol. She said:

In the course of my duty I have examined patients under the influence of alcohol countless times. I have not given evidence in court on this aspect. I can testify on the effect of alcohol. I say that the accused was under the influence of alcohol because, firstly, admission by the accused himself that he had consumed alcohol before the incident incurred, secondly, when the accused was brought in several factors clinically indicated that he was alcohol intoxicated. Firstly, the alcohol smell in his breath. Secondly, his flushed face. Thirdly, his staggering gait. Fourthly, the unsteadiness of his hand. Fifthly, his slurred speech. Sixthly, extreme state of agitation. All these factors point towards alcohol intoxication. He was lucid. I say this because the accused was able to state his full name clearly and was able to write P17 without difficulty and Prof Dr Ernest Yeoh had just joined the Emergency Department prior to the incident. This means that the accused is able to recall and is orientated to time, place and person when I saw him at 1.15 am.

In her cross-examination, PW8 explained what she meant when she said the accused was 'restless'. As she said:

By the accused being 'restless' I mean he was agitated, moving about and trying to get up from the chair and trying to get the police to remove the handcuffs. He asked the police to remove the handcuffs. There was no response from the police. They just pushed him on to the chair. The police were ensuring that the accused was seated except during my physical examination of the accused when I had to ask him to stand up.

She said that the accused was not aware as to why the police had arrested him. In explaining the consent given by the accused for the taking of his blood and urine samples, she said:

I obtained consent from the accused for the samples to be taken. It is in a form brought by the police. The form was handed to the police. The accused signed the consent form in front of me and the police. ... There is no copy of the consent in my file. The consent was an intelligent consent given by the accused.

He signed the consent in front of me. I am absolutely certain. I did not tell the accused that he had the right to refuse to give samples. The consent form is police type written form in this type of cases. It is only consent to

- A** take blood and urine. I deny that the form was re-signed by the accused. I am aware of this.
- Under further questioning, she explained the symptoms of hypoglycaemia. She also said that she did not do any clinical tests to determine the intoxication of the accused. She agreed that the accused's pulse rate of 102
- B** is consistent with hypoglycaemia amongst other things. She added that it is possible for a patient who is regarded as being under the influence of alcohol to be going through a hypoglycaemia episode. In answer to further questions, she said:
- When I examined the accused, I found him to be alcohol intoxicated based on my clinical findings. It is possible for him to be hypoglycaemia.
- C** In commenting on the state of mind of the accused at 8pm, she said:
- I cannot attest to the accused's state of mind at the time of the incident. I can only say that I saw him five hours after the incident. It is possible that he was coming out of his condition at 8pm. I can only testify positively on this if I knew his condition at 8pm. I cannot attest to the behaviour of the accused at 8pm as I was not there. If his condition at 8pm was under alcohol then after five hours it would have worn off a little. If it was alcohol his condition at 8.00 pm would have been more severe. The level of intoxication at 8.00 pm would be more. The outward symptoms of intoxication would be more.
- D**
- She was re-examined on the pulse rate of 102 of the accused. She said:
- E** A pulse rate of 102 means that it is slightly higher. There are hundreds of causes for this rate. Anger can be a cause. This pulse rate is not conclusive of hypoglycaemia.
- In explaining whether the accused had presented himself as a case of hypoglycaemia, she said:
- F** In my opinion, the accused did not present himself as a hypoglycaemia patient. I say this because at that time the accused was alert, was not sweating, was able to answer my questions. It is possible that prior to my seeing him, he had recovered from hypoglycaemia. There will be no symptoms to indicate that a person has recovered from hypoglycaemia.
- G** PW13 is a chemist attached to the Department of Chemistry, Petaling Jaya. The relevant exhibits in this case were handed to him by PW15 for examination. He examined a Proton Perdana motor car bearing registration number BFW 6366. He found that there was a hole on the offside region of the windscreen of the car. It was consistent with being a bullet hole. His examination of landrover bearing registration number ZB 4970 revealed a penetrating hole on the nearside rear door frame consistent with being a bullet hole. He also found a penetrating hole in the backrest of the rear passenger seat which was consistent with being a bullet hole. There was also a non-penetrating hole on the front of the backrest of the rear passenger seat which was consistent with having been hit with a bullet. PW13 analyzed the
- H**
- I** other exhibits handed to him. He found exh P9 to have gunpowder residues in its barrel indicating that it had been fired since it was last cleaned. He also found that exhs P54, P55, P28, P32 and P34 were fired from exh P9. He

analyzed the blood sample in exh P19 and found it to contain 198mg of ethyl alcohol per 100ml and the urine sample in exh P20B to contain 257mg of ethyl alcohol per 100ml. In commenting on the blood and urine samples, he said:

A

With regard to the blood and urine specimens that I received, the quality was good for me to carry out analysis of the alcohol content on them. The results of the alcohol in the urine as compared to that in the blood specimen was approximately in the ratio of four to three. This is consistent with the ratio normally reported to show the distribution of the alcohol in the urine and the blood. This level of alcohol in the urine and blood will have some effects on the person.

B

He found gun shot residues on the receptacle labelled Left Web ('exh P41'). In commenting on this finding, he said:

C

The finding of gunshot residue on the receptacle labelled Left Web does not necessarily show that the weapon was held in the left hand because in a living person the activities after a shooting incident may cause a re-distribution of the gunshot residue between the two hands.

D

PW13 was cross-examined on the integrity of the blood and urine samples he analyzed. In answer to questions, he said:

The integrity may depend on how the samples were kept and preserved. Ideally blood and urine samples should be kept in the refrigerator. When these samples reached me they came in an envelope. The blood and urine samples arrived at my office at 2.44pm on 24 August 2000. I have no idea how it was kept by the police. I have no idea how the samples were prepared and contained in the receptacles. When I carried out the actual analysis the samples showed that they were good.

E

The method of analysis I used for both the blood and urine samples I used the gaschromatography method to determine the content of alcohol. This is the accepted method for this test. Before I subjected the samples to this test I did a physical examination of the two samples to determine whether the samples were still good. After I received the samples I kept them in the refrigerator.

F

In answering questions on gunshot residue found on hands, PW13 said:

G

When one is shooting with a pistol, the gunpowder will fall on the back of the hand and the palm is protected. This may be true if one hand is used and the other hand is far away. If both hands are used the back of both the hands are exposed to gunpowder.

The presence of gunshot residue on the hands, whether left or right can be used to indicate that a person could have been involved in firing a weapon recently. But one has to be very cautious in interpreting the deposition of the gunshot residue on a specific hand to link it with the hand that was used to shoot. This is because in the case of a living person, his subsequent activities after the shooting incident may result in the loss or subsequent re-distribution of the gunshot residue originally deposited. It is possible that the hand where the gunpowder is found is the hand that fired the shot. This possibility is not necessarily strengthened by the fact that nothing was found on the other hand.

H**I**

- A** PW13 also said that when a person has taken alcohol, there will be a period called the peak period. The period peaks for a while and then drops. This is true in both urine and blood samples. He said that by looking at the ratio of the urine and blood results, one can say whether the samples were taken at the peak period or not. He also said that it is possible to estimate the alcohol level in a person for a certain period prior to the taking of the samples. He added:

It is by no means accurate because in doing this estimation, we have to assume certain variables. We will never know the variables absolutely. The more we fix the variables then the estimation is more reliable. The variables include body weight. The number of times that a person has urinated prior to the taking of the specimen can be a variable for the urine test but not the blood test.

- C**
- D** At the close of the case for the prosecution, the learned deputy public prosecutor offered to the defence Hassan bin Ujang, Mohd Rapet bin SM Abdul, Wan Abdul Ghani, Shaari bin Salleh, Mat Yusoff bin Hamat, R Subramaniam, Ghazali Abdullah, Noor Salasawati, Lam Thong Soon, Chong Chee Hong, Mohd Ali bin Mohd Salleh, How Yang Yu, Raja Mohd Azizul, Ooi Mung Lern, Zulkarnain Abu Bakar, Chan Kok Heng, Yew Yong Fei, Tan Ah Chai, Chief Inspector Abdul Rahim, Sjn Ang Choo Yu, Lance Corporal Selvakumar, Inspector Said Azizi and ASP Thomas Woon to the defence. The defence required Noor Salasawati, Lim Thong Soon, Chong Chee Hong, Mohd Ali bin Mohd Salleh, How Yong Yu, Raja Mohd Azizul, Ooi Mun Lern, Zulkarnain Abu Bakar, Chan Kok Heng, Liew Yong Fei, Tan Ah Chai, Chief Inspector Abdul Rahim, ASP Thomas Woon and Dato' Abdul Aziz (not offered by the prosecution).

- E**
- F** In his submission, Dato' Muhammad Shafee Abdullah, leading counsel for the accused, outlined the facts of the case and said that they did not indicate any intention on the part of the accused to commit the offence. He said that cl (d) of s 300 of the Penal Code was inapplicable as it applies only in cases envisaged by illustration (d) of the section. Encik Abdul Karim bin Abdul Jalil, the deputy public prosecutor leading the prosecution, referred to the facts of the case and said that the prosecution had made out a prima facie case.

- G**
- H** In determining whether the prosecution had made out a prima facie case, it was first necessary to consider the effect of intoxication in criminal liability as the accused appeared to have been under the influence of alcohol at the time of the incident. The law relating to intoxication in criminal law is contained in ss 85 and 86 of the Penal Code ('s 85 and s 86'). Both the sections read as follows:

Section 85

- I**
- (1) Save as provided in this section and in s 86, intoxication shall not constitute a defence to any criminal charge.
 - (2) Intoxication shall be a defence to any criminal charge if by reason thereof the person charged at the time of the act or omission complained of did not know that such act or omission was wrong or did not know what he was doing and —

- (a) the state of intoxication was caused without his consent by the malicious or negligent act of another person; or
- (b) the person charged was by reason of intoxication insane, temporarily or otherwise, at the time of such act or omission.

Section 86

- (1) Where the defence under sub-s 85(2) is established, then in a case falling under paragraph (a) thereof the accused person shall be acquitted, and in a case falling under paragraph (b), the provisions of s 84 of this Code, ss 347 and 348 of the Criminal Procedure Code shall apply.
- (2) Intoxication shall be taken into account for the purpose of determining whether the person charged had formed any intention, specific or otherwise, in the absence of which he would not be guilty of the offence.
- (3) For the purpose of this and the preceding section 'intoxication' shall be deemed to include a state produced by narcotics or drugs.

It will be observed that the two sections, contemplating different levels of intoxication (see *Public Prosecutor v Teo Heng Chye* [1989] 3 MLJ 205), are designed to meet three different situations. Firstly, by virtue of s 85(2)(a) intoxication is a defence to any criminal charge if by reason of the intoxication, the accused at the time of the act or omission complained of, did not know that such act or omission was wrong or did not know what he was doing and his state of intoxication was caused without his consent by the malicious or negligent act of another person. In this situation, the accused is entitled to an acquittal. Secondly, by virtue of s 85(2)(b) where the accused is by reason of intoxication rendered insane, temporarily or otherwise at the time of his act, he is entitled to a special acquittal by reason of insanity. The intoxication could even have been caused voluntarily. In the event of a finding under this sub-section the accused will be dealt with under s 84 of the Penal Code and ss 347 and 348 of the Criminal Procedure Code. Thirdly, s 86(2) provides that where the accused is intoxicated, voluntarily or not, his intoxication is to be taken into account for the purpose of determining whether he had formed any intention, specific or otherwise. The sub-section does not refer to other mental elements such as knowledge or rashness. In *Director of Public Prosecutions v Majewski* [1977] AC 443, the House of Lords has confirmed the rule that evidence of self-induced intoxication negating mens rea is a defence to a charge of a crime requiring a specific intention but not to a charge of any other crime. It follows that s 86(2) will have no application in the case of offences requiring such other mental elements. Thus, as observed by Yong Pung How CJ in *Juma'at bin Samad v Public Prosecutor* [1993] 3 SLR 338 at p 344–345:

It is to be noted that an anomalous consequence of the drafting in s 86(2) is that it applies only where the mens rea for an offence is intention, in contradistinction to offences requiring other forms of mens rea specified in the Penal Code, for example, knowledge or rashness. The result is somewhat disturbing, for example s 86(2) would apply to a charge of murder under s 300(a), (b) or (c) but not to a charge of murder under s 300(d). However, the words of the provision are clear and the consequences though discomfiting are not of such degree of absurdity as would justify the court departing from a literal interpretation.

- A This view is logical in view of the higher level of mental activity required to form an intention as contrasted with, say, knowledge. As the *Penal Law of India* by Dr Sir Hari Singh Gour (11th Ed) Vol V says at p 2427:

It can be seen that ‘knowledge’ as contrasted with ‘intention’ signify a state of mental realization with the base state of conscious awareness of certain facts in which human mind remains supine or inactive. On the other hand, ‘intention’ is a conscious state in which mental faculties are aroused into activity and summoned into action for the purpose of achieving a conceived end. It means shaping one’s conduct so as to bring a certain event. Therefore, in the case of ‘intention’ mental faculties are projected in a set direction.

- C I also refer to *Emperor v Mt Dhirajia* AIR 1940 All 486 where Braund J said at p 488:

We regret that we are unable to go as far as this. ‘Intention’ appears to us to be one thing and ‘knowledge’ appears to us to be a different thing. In order to possess and to form an intention there must be a capacity for reason. And when by some extraneous force the capacity for reason has been ousted, it seems to us that the capacity to form an intention must have been unseated too. But to our minds, knowledge stands upon a different footing. Some degree of knowledge must, we think, be attributed to every sane person. Obviously, the degree of knowledge which any particular person can be assumed to possess must vary. For instance, we cannot attribute the same degree of knowledge to an uneducated as to an educated person. But we think that to some extent knowledge must be attributed to everyone who is sane. And what we have to consider here is whether it is possible for us — treating Mt Dhirajia as a sane person, which we are bound to do — to conclude that she could possibly have been ignorant of the fact that the act of jumping into a well with a baby in her arms was likely to cause that baby’s death. We do not think we can.

- Where, therefore, s 86(2) is rendered inapplicable in a particular case by virtue of knowledge only being the ingredient to be proved it must be taken to mean that an intoxicated person had the same knowledge as he would have had if he had not been intoxicated. This is the inevitable result as the exclusion of intoxication as a factor to be considered in determining liability simply means that it warrants no consideration at all. Of course, if the level of intoxication of a person is so high that he did not know that what he was doing is wrong or did not know what he was doing, the burden is on him to bring his case within the meaning of s 85(2)(b). Where s 86(2) applies, the accused would not be guilty of the offence. However, in such a situation, the accused may be found guilty of a lesser offence where intention is not in issue (see *Juma’at bin Samad v Public Prosecutor*; *Suba Singh v Public Prosecutor* [1962] MLJ 122; *Tan Hung Song v Rex* [1951] MLJ 181; *Public Prosecutor v Teo Heng Chye*).

- I The resultant matter for consideration is the question of burden of proof with regard to intoxication within the meaning of s 86(2). In dealing with this issue, Yong Pung How CJ posed this question in *Juma’at bin Samad v Public Prosecutor* at p 345:

Another aspect of s 86(2) on which there is some controversy is whether the burden of proof falls on the accused person to prove on a balance of probabilities that he was so intoxicated that he did not form the necessary intention, or whether the burden remains on the prosecution to prove beyond reasonable doubt that, in spite of the intoxication, the accused person did form the requisite intention.

And answered it in this way:

I am inclined to favour the former view. Sections 85 and 86 are found in Ch 4 of the Penal Code which deals with general exceptions, for which the burden of proof falls on the accused persons by virtue of s 107 of the Evidence Act (Cap 97). Furthermore, s 85(1) expressly refers to s 86 as dealing with the 'defence' of intoxication. Therefore, any evidence of intoxication does not affect the prosecution's case; in proving beyond reasonable doubt that the accused had the necessary mens rea the prosecution is entitled to treat the accused as if he were sober. The court may no doubt have to answer some rather hypothetical and artificial questions in the process, but this is preferable to a solution which is completely out of accord with the general scheme of the Penal Code and the Evidence Act. Furthermore, the scope of s 86(2) generously extends to voluntary intoxication, a legal excuse which, in my view, can never put an accused in a more favourable position than another accused who pleads any of the other defences.

This view has been followed consistently in Singapore (see, for example, *Suradet v Public Prosecutor* [1993] 3 SLR 265; *Indra Wijaya Ibrahim v Public Prosecutor* [1995] 2 SLR 42; *Sim Eng Teck v Public Prosecutor* [1998] 3 SLR 818 and *S Nagarajan s/o Kuppusamy v Public Prosecutor* [1999] 2 SLR 111). Thus, the burden of proof to establish intoxication is on the accused. This is in line with the provisions of s 105 of the Evidence Act 1950. It is settled law that where a burden is placed on an accused person it can be discharged by evidence of witnesses for the prosecution as well as witnesses for the defence (see *Public Prosecutor v Mohamed Noor Jantan* [1979] 2 MLJ 80; *Wong Chooi v Public Prosecutor* [1967] 2 MLJ 180). This is particularly significant in the case of s 86(2) which requires intoxication to be taken into account for the purpose of determining the existence of intention for which a ruling must be made at the close of the case for the prosecution. In an appropriate case, a finding on intoxication can therefore be made at the close of the case for the prosecution.

In dealing with the manner of making a ruling on intoxication useful, reference may be made to *R v Sheehan* [1975] 2 All ER 960 where Geoffrey Lane LJ said at p 964:

Indeed, in cases where drunkenness and its possible effect on the defendant's mens rea is in issue, we think that the proper direction to the jury is, first, to warn them that the mere fact that the defendant's mind was affected by drink so that he acted in a way in which he would not have done had he been sober does not assist him at all, provided that the necessary intention was there. A drunken intent is nevertheless an intent. Secondly, and subject to this, the jury should merely be instructed to have regard to all the evidence, including that relating to drink, to draw such inferences as they think proper from the evidence, and on that basis to ask themselves whether they feel sure that at the material time the defendant had the requisite intent.

A It is now appropriate to consider the requirement of intention as an element to be proved by the prosecution in the offence with which the accused has been charged so as to determine whether s 86(2) is to be activated. The charge of murder faced by the accused is defined in s 300 of the Penal Code ('s 300'). It reads as follows:

- B** Except in the cases hereinafter excepted, culpable homicide is murder —
- (a) if the act by which the death is caused is done with the intention of causing death;
 - (b) if it is done with the intention of causing such bodily injury as the offender knows to be likely to cause the death of the person to whom the harm is caused;
 - C** (c) if it is done with the intention of causing bodily injury to any person, and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death; or
 - (d) if the person committing the act knows that it is so imminently dangerous that it must in all probability cause death, or such bodily injury as is likely to cause death, and commits such act without any excuse for incurring the risk of causing death, or such injury as aforesaid.
- D**

It will be observed that cll (a), (b) and (c) of s 300 contemplate intention in one form or another. In cl (a), it is intention to cause death. In cll (b) and (c), it is intention to cause bodily injury. On the other hand, there is no requirement of an intention to cause death or bodily injury in cl (d) of s 300 ('cl (d)'). Thus, intention is not a necessary element in cl (d); all that is required to be proved is knowledge that the act is likely to cause death (see *Inder Singh v Crown* (1928) ILR 10 Lah). It has been held that this clause is intended to apply only when there is no intention to cause death, or in other words, when the three earlier clauses are inapplicable (see *Hasta Ismail v Emperor* AIR 1937 Lah 593; *Behari v State* AIR 1953 All 203). Section 86(2) is therefore applicable to cll (a), (b) and (c) of s 300 and inapplicable to cl (d).

Having dealt with the law, I shall now consider:

- G** (a) the state of intoxication of the accused at the material time so as to determine whether he had formed the requisite intention; and
- (b) the criminal liability of the accused if it is found that he did not form the requisite intention by virtue of his intoxication.

I pause to add that intoxication was not specifically raised by the accused as a defence at this stage. However, it is the duty of the court itself to consider a defence if the evidence adduced discloses it even if it has not been raised (see *Chan Chor Shuh v Public Prosecutor* [2003] 2 MLJ 26; *Abdullah bin Jacomah v Public Prosecutor* [2002] 6 MLJ 324).

The state of intoxication of the accused

- I** The behaviour of the accused leading to the shooting of the deceased and thereafter can be described as abnormal. I have described this as testified by the various witnesses in an earlier part of the judgment. His car was parked

on the middle of the road in the face of oncoming traffic. When the motorcyclist whom he had stopped sped off, he fired two shots in the air. He stopped PW4 by pointing his gun. When PW4 obeyed his instructions he was satisfied. It must be observed that the deceased was not known to the accused previously and he fired the shot merely because his instructions were not obeyed. When the deceased did not respond to him, he fired a shot at her. This cannot be described as normal conduct. The behaviour of the accused after firing the shot is significant. An attempt by the accused to get away from the place of incident may negate a state of intoxication as illustrated by *Basdev v State of Pepsu* AIR 1956 SC 488. But in this case, the accused made no such attempt. On the other hand, when PW6 shouted, the accused fired shots in his direction. He then fired a shot in the direction of a parked military landrover and walked down the road and sat on the road for a while. Whenever people tried to approach him, he pointed his gun at them. PW4 said that the accused looked normal and was steady and not staggering when walking. PW6 said that the behaviour of the accused gave him the impression that he was confused. PW6 also said that the accused was walking aimlessly as if he was lost and appeared to be unconscious of his surroundings. PW7 said that the accused was steady prior to his arrest. While at the police station, the accused slept off and passed urine and faeces in his pants. PW15 said that he did not caution the accused as he was under the influence of alcohol and was aggressive. The accused used vulgar words to find out why he was arrested. The accused also referred to PW15 as Khoo Ah Si. PW8 said that clinically, the accused was under the influence of alcohol though he was alert. She said that the accused had smell of alcohol in his breath; his face was flushed; he had a staggering gait; his hand was unsteady; he had slurred speech and was in a state of extreme agitation. The accused had consumed beer prior to the incident. The blood sample of the accused was found to contain 198mg of ethyl alcohol per 100ml and the urine sample contained 257mg of ethyl alcohol per 100ml. PW8 said that the level of alcohol in the accused at 8pm would have been more.

The evidence of PW8 that the level of alcohol in the accused would have been more than what was determined from the blood and urine samples taken from him mean that the levels would have been higher at the time of the incident. There is no evidence to indicate what that level would have been. However, it must be observed that the blood alcohol level itself is not conclusive to determine the degree of intoxication of a person. As Chao Hick Tin JC said in *Public Prosecutor v Ramasamy a/l Sebastian* [1991] 1 MLJ 75 at p 81:

In any event, from the evidence of the experts, the blood alcohol level itself can never be conclusive to determine the degree of intoxication of the accused. Different people react differently to the same blood alcohol level. It makes a great difference whether the person is or is not an experienced drinker. In our judgment, and here we accept the opinion of Dr Chan (PW15), the more reliable indicator of the state of mind of the accused must be the conduct of the accused immediately prior to and after the offence.

In *Public Prosecutor v Arwang Raduan bin Arwang Bol* [1998] 5 MLJ 460, Tee Ah Seng J said at p 477:

- A** In the absence of expert evidence, I am of the view that the more reliable indicator of the state of the mind of the accused must be the conduct of the accused immediately prior to and after the offence.

- The recent judgment of the Court of Appeal in *Francis all Anthonysamy v Public Prosecutor* [2003] 2 MLJ 49 supports the proposition that the defence of intoxication can be established without the support of scientific evidence.
- B** Thus, what is more pertinent is evidence of the state of mind of the person at the material time. The best evidence to establish his state of mind is his conduct prior to, at the time of, and after the offence. I say this because what is in issue is the effect of intoxication on the state of mind of the person in order to determine whether he could have formed the necessary intention.
- C** This is best reflected by his conduct and not the level of his intoxication. The blood alcohol level is certainly an element, and an important one, to explain his conduct. The significance of the conduct of a person as an indicator of his level of intoxication is illustrated by the Federal Court case of *Liew Koh Tai v Public Prosecutor* [1965] MLJ 54 where, on the facts of the case, the conduct of the accused was held to negate intoxication.
- D** In *Rajeevan Edakalavan v Public Prosecutor* [1998] 1 SLR 815, the rational behaviour of the accused coupled with low blood alcohol level was held to be unsustainable to support the plea of intoxication. In *Tan Hung Song v R*, it was recognized that a blood alcohol reading of 215mg of ethyl alcohol to 100ml of urine shows that a person was in a certain state of intoxication.
- E** In *Public Prosecutor v Tan Ho Teck* [1988] 3 MLJ 264, part of the evidence that established the heavy intoxication of the accused was the fact that his blood was found to contain 232mg alcohol per 100ml of blood.

- However, it has been recognized that the mere fact that a person is intoxicated does not preclude him from forming an intention (see *Aladin v Public Prosecutor* [1964] 1 MLJ 345). As Stephone J said in *R v Doherty* 16 Cox CC 306 at p 308:
- F**

A drunken man may form an intention to kill another, or to do grievous bodily harm to him, or he may not, but, if he did form that intention, although a drunken intention, he is just as much guilty of murder as if he had been sober.

- G** A drunken man is presumed to have the capacity to form the specific intent necessary to constitute the crime, unless evidence is given from which it can reasonably be informed that he was incapable of forming it (see *Kennedy v HM Advocate* (1944) SC (J) 171; *Bratty v Attorney-General for Northern Ireland* [1963] AC 386). The behaviour of the accused in this case prior to the incident, the abnormal manner in which the deceased was shot, his behaviour subsequent to the incident and the evidence of PW8 who said that the accused was under the influence of alcohol when she examined him based on her clinical findings and her evidence that the blood alcohol level in the accused at 8pm would have been more than 198mg of ethyl alcohol per 100ml compelled me to conclude that his intoxicated state was weighty enough to preclude him from forming the necessary intention in respect of
- H**
- I** the offence with which he had been charged. I was therefore satisfied that the conduct of the accused in firing the shot which killed the deceased militated against a finding that it was done so intentionally as required by

cll (a), (b) or (c) of s 300 pursuant to the application of s 86(2) to the facts of the case. I was therefore of the opinion that the case against the accused did not come within the ambit of these three clauses. The state of mind of the accused certainly did not fall within the description of intoxication in s 85(2)(b).

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The criminal liability of the accused under cl (d)

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That brought into focus the liability of the accused under cl (d). It must be observed that this clause is not intended to apply to cases in which a person intends to inflict an injury likely to cause death because it speaks of knowledge and not of intention of an injury likely to cause death (see *Behari v State*). Thus, as I said earlier, s 86(2) is inapplicable to this clause. A matter of immediate concern, bearing in mind the submission advanced by the defence, is the circumstances in which it can be invoked. The defence contended that it is confined in its operation to instances similar to that contained in illustration (d) of s 300. It reads as follows:

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A without any excuse fires a loaded cannon into a crowd of persons and kills one of them. A is guilty of murder, although he may not have had a premeditated design to kill any particular individual.

D

This refers to a situation where the act of the accused is directed against no one in particular. The issue for determination is whether this clause applies only to cases envisaged by the illustration. It must first be observed that illustrations to a section are not an exhaustive explanation of the operation of the section. They are merely useful guides to ascertain the meaning of a section (see *Munah binti Ali v Public Prosecutor* [1958] MLJ 159; *Ramamoorthy v Public Prosecutor* [1948] MLJ 43). They cannot therefore be construed as constituting the section itself thereby controlling its whole meaning (see *Purshoftam Ishvar Amin v Emperor* AIR 1921 Bom 3). Thus, in commenting on the clause *The Penal Law of India* by Dr Sir Hari Singh Gour (11th Ed) Vol 3 says at p 2460:

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It has been stated before that this clause was intended to meet a case where the act of the accused is directed against no one in particular (see s 299). But this is by no means necessary, nor, indeed should the clause be held confined only to such cases. For, there are cases in which a single individual may be the target of the accused's malice as in the case of the mother exposing her illegitimate child in the hope that it may be picked up but with the certain knowledge that its exposure will in all probability compass its death.

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In considering the extent of the clause the Indian Supreme Court said in *State of Madhya Pradesh v Ram Prasad* AIR 1968 SC 881 that although it is usually invoked in those cases where there is no intention to cause the death of any particular person, as shown by the illustration, it may on its own terms be used in those cases where there is such callousness towards the result and the risk taken is such that it may be stated that the person knows that the act is likely to cause death or such bodily injury as is likely to cause death. In that case, it was held that the case of the accused fell within

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- A cl (d) when the accused's mistress died as a result of her dress being set on fire by him. In *Gyarsibai v State* AIR 1953 MB 61, there were constant quarrels between the accused and her sister-in-law. In one of the quarrels, the accused was asked to leave the house. Thereupon, she took her three infant children and jumped into a well with them. She survived but the children died. It was held that it cannot be said that the accused did not
- B have the knowledge that her act was so imminently dangerous as to cause the death of her children; it cannot be said that she was in such an abnormal state of mind that she could not have any knowledge of the nature of her act; the fact that the quarrels between the accused and her sister-in-law had made her life become unbearable cannot be regarded as a valid justification for her act of jumping into a well with her children. Accordingly, she was
- C found guilty of murder under cl (d). Be that as it may, and in any event, in this case the act of the accused was not directed specifically at the deceased in particular as any other person similarly circumstanced as she would have suffered the same fate. I was therefore satisfied that the facts of this case are capable of coming within the ambit of cl (d) subject, of course, to fulfilment
- D of the requirements of the clause.

Clause (d) requires proof of the following:

- (a) the person committing the act must know that it is so imminently dangerous that it must in all probability cause death, or such bodily injury as is likely to cause death;
- E (b) the act must have been committed without any excuse for incurring the risk of causing death, or such injury.

I considered whether the two ingredients have been proved in order to determine whether the prosecution had made out a prima facie case against the accused.

F *Ingredient (a)*

- The knowledge which accompanies the act must be that the act was so imminently dangerous that it must in all probability cause death or such bodily injury as is likely to cause death. Knowledge is an awareness of
- G the consequences of an act (see *Basdev v State of Pepsu* AIR 1956 SC 488). It is a mental act and must be inferred from the facts and circumstances of a particular case. In dealing with the manner of ascertaining knowledge *The Penal Law of India* by Sir Hari Singh Gour (11th Ed) Vol 3 says at p 2381:

- H Criminal knowledge, is then, in such cases demonstrated a posteriori. It takes into account not only knowledge but means of knowledge, not only the knowledge which is, but which, judging from the effect, ought to have been in the accused. A person may then truthfully declare that he did not know that his act was likely to cause death and yet he may be rightly found to have had that knowledge. The truth is that in civil cases arising out of tort as well as in
- I criminal cases, the standard which the court fixes before itself is that of a reasonable man and the question it ultimately asks itself is, not whether the accused had the knowledge, but whether as a reasonable man he could have had that knowledge. And for this purpose, the act itself is the real test.

The nature of the weapon used is a matter to be considered to determine the state of mind of a person at the time of the offence. As *The Penal Law of India* by Sir Hari Singh Gour (11th Ed) Vol 3 says at p 2387:

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It has been said that in inferring knowledge the court looks to the result. If it is one which could not have been arrived at without fore-knowledge, the court presumes it. Such knowledge may be legitimately presumed where the assault is committed with an axe or a dao or other deadly weapon, or where a man is hit with great force on a vital part of his body.

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In commenting on the words 'imminently dangerous' in cl (d) *The Penal Law of India* by Dr Sir Hari Singh Gour (11th Ed) Vol 3 says at p 2459:

The question whether an act is imminently dangerous depends upon the nature of the act and its evident risk to human life. The word 'imminently' implies a risk which is both threatening and near.

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As Modi J said in *Janardan Prasad v State of Rajasthan* 1977 Cr LJ 68 at p 72:

The earlier part of this clause refers to the cases where the act of the accused is itself so imminently dangerous that it must, in all probability, cause death or such bodily injury as is likely to cause death. The emphasis in the preceding part of cl 4 is on the imminently dangerous nature of the act itself.

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I then considered the facts of the case in order to determine whether the accused had the required knowledge. When the motorcyclist sped off, he fired two shots in the air. When he stopped PW4, he did not fire as PW4 had followed the instructions that were given. He then knocked at the windscreen at the driver's side of the window of the Perdana asking the driver to come out. He only fired after the person refused to open the door of the car. It was a response to his orders not being obeyed. The shot was fired at point blank range at the front windscreen of the car. If the door had been opened the accused would not have fired the shot just as in the case of PW4. He was in a position to decide when to fire and when not to. He was therefore conscious of what he was doing. The conduct of the accused in firing the shot shows that it was done with absolute callousness towards the result. It was in general disregard for human life and safety. This clause is usually applied in such cases (see *Janardan Prasad v State of Rajasthan*). The use of a firearm by the accused is significant. It is a lethal weapon. Where a shot is fired at a person there is a strong probability that it will cause death or such bodily injury as is likely to cause death. This is particularly so in this case bearing in mind the circumstances in which the shot was fired at the deceased. The shot was fired from outside at the deceased who was seated inside the car. Obviously, it will hit the upper part of the body of the deceased where the vital organs are. Accordingly the bullet fired by the accused hit the chest area of the deceased and PW16 said that the injuries sustained were necessarily fatal. Firing at a person at close range with a pistol in the circumstances as in this case is an evident risk to human life. It is a risk which is both threatening and near. It amounts to an act which is so imminently dangerous that it must, in all probability, cause death or such bodily injury as is likely to cause death. As Wanchoo CJ said in *Kanji v State* (1953) Cr LJ 434 at p 435:

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- A** We are, therefore of the opinion that where a person takes the risk of shooting at another the act would ordinarily be an imminently dangerous act which must in all probability cause death or such bodily injury as is likely to cause death and the case would be covered by the fourth clause in s 300.
- It can be presumed that a person had knowledge of the danger of his act (see *Bhagat Singh v Emperor* 121 IC Lah 726; *Ranji v State* 1952 RLW 145). As
- B** Dixit J said in *Gyarsibai v State* at p 62:
- Every sane person — and in this case we are bound to take it that the appellant was sane — is presumed to have some knowledge of the nature of his act. This knowledge is not negated by any mental condition short of insanity.
- C** An act which is necessarily fatal carries with it knowledge of its consequences (see *Emperor v Mt Dhirajia*). Thus, no special knowledge is needed to know that one may cause death or such bodily injury as is likely to cause death by shooting as in the case of setting fire to the clothes of a person (see *State of Madhya Pradesh v Ram Prasad*). Similarly, it has been held that the act of an accused in jumping into a well with her children is
- D** clearly one done by her knowing that it must in all probability cause the death of her children (see *Emperor v Mt Dhirajia*). This presumption may of course be rebutted. In my opinion, the degree of knowledge that is required can therefore be fastened on the accused from the imminently dangerous nature of the act itself of shooting at the deceased at point blank range based on a decision made for that purpose. As I have explained earlier, the fact
- E** that he was in a state of intoxication does not exclude him from liability under cl (d). In *Kanji v State*, the accused who was heavily drunk shot at a boy with a gun from a distance of about ten paces and the shot struck the abdomen of the boy who died shortly afterwards. It was held that the accused was guilty of murder under this clause. In *Dil Mohammad v State*
- F** (1941) 21 Pat 250 where the injury inflicted by the accused person in a state of intoxication was of such a nature as any sober man must surely have known that the injury was likely to cause death and where the injury was sufficient in the ordinary course of nature to cause death, it was held that although the act was not intended to cause death, the offence was one of murder. In *Pal Singh v State* AIR 1917 Lah 226, it was held that where the
- G** accused persons who were under the influence of liquor assaulted the deceased and literally beat him to death with *lathis* without any direct motive they were guilty of murder as they must have known that their act was so imminently dangerous that it must in all probability cause death or such bodily injury as is likely to cause death. Needless to say, if the level of intoxication of the accused was so high that he did not know that what he
- H** was doing was wrong or did not know what he was doing the burden is on him to bring his case within the meaning of s 85(2)(b). I was therefore satisfied that the prosecution had established this ingredient.

Ingredient (b)

- I** In order for there to be an offence under cl (d), the act must have been wholly inexcusable. In commenting on this ingredient, *The Penal Law of India* by Dr Sir Hari Singh Gour (11th Ed) Vol 3 says at p 2458:

The expression 'without any excuse' means without any exculpatory circumstances other than those mentioned in the five exceptions to this section. The word 'excuse' here means something which is a justification of the act and does not merely mitigate the crime.

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In *Emperor v Mt Dhirajia*, a woman was ill-treated by her husband. There was a quarrel between the two and the husband had threatened that he would beat her. During the night, the woman took her six-month old baby and went away from the house. When she suddenly saw her husband behind her, she got into a panic and jumped down a well nearby with the baby in her arms. The baby died and the woman recovered. She was charged with the murder of the child. It was held that an intention to cause the death of the child could not be attributed to the woman, though she must be attributed with the knowledge — however primitive or frightened she must have been — that such an imminently dangerous act as jumping down the well was likely to cause the child's death but the culpable homicide did not amount to murder because, considering the state of panic she was in, there was excuse for incurring the risk of causing death within the meaning of cl (d). In this case, the accused was not in the plight of the woman as in the case that I have just referred to. He shot at the deceased when she refused to open the door of her car and had absolutely no reason to do so. In such circumstances, the firing of the shot at her is wholly inexcusable both on legal and moral grounds. It does not fall within any of the exceptions enumerated in s 300. I was therefore satisfied that the prosecution had established that the act of the accused was wholly inexcusable.

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Thus, the prosecution had made out a prima facie case against the accused as defined in cl (d). I then called upon him to enter his defence. He elected to give evidence on oath.

The accused was the executive director of Green Mountain Holdings (M) Sdn Bhd. On 22 August 2000, he got up late in the morning. He is not a person who can get up easily. Normally, his wife Noor Salasawati bte Mohd Taib ('DW4') 'yanks' him out of bed if he has an urgent appointment. DW4 prepares his breakfast consisting of milo with condensed milk and a teaspoon of glucose and toasted bread with peanut butter. As the accused said:

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The glucose is added on doctor's recommendation as I am supposed to have low blood sugar. I have been advised by doctors to take glucose about ten years prior to the incident. The doctors who advised me are Dato' Dr Ridzwan Bakar and Dr Thiagi.

On the morning in question, the accused drove himself to his office in his Volvo Stationwagon 850 TS bearing registration number WS 9 for the meeting. He arrived in the office after 11am. The Board meeting then commenced. The accused said:

H

The persons who attended are Mr William Chang, Mr Lim Thang Soon, Mr Tan Ah Chai and Mr Liew Yong Fee. They are all directors of the company. I was also a director of the company. There wasn't good news at the meeting on the progress of the company. There was discussion on the problems faced by the company in Kuala Lumpur as well as in China. Funds were not coming

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- A** in as expected for the China project. There was another problem not related to the project as such. It was in relation to one of the directors. It was a conflict of interest problem with the company. These were the two major problems that morning. The meeting was over at about 1.45pm.

- B** After the meeting, all of them except for Mr Liew Yong Fee went for lunch to a restaurant which they frequent. The accused calls the restaurant Ah Ming's place. They ordered food consisting of Soon Hock fish, vegetables, omelette with prawns and onions, rice, Chinese tea and beer. The general mood at the lunch was one of disappointment with the way things were progressing in the company. The accused took some glasses of Tiger beer. With regard to his drinking habits, he said:

- C** My normal capacity for beer is quite low. It is about six glasses. It is a bulky and gassy drink. So my maximum is about six glasses. If I had a choice of drinks to take for lunch I would take beer. In the evening, it is normally whisky. After a game of golf also I take beer. With a Western dinner when wine is served, I would complement it with brandy.

- D** He has had experience of being drunk in his younger days. As he said:
I would normally go off to sleep.

The accused said that from the point of time he had lunch, he could not remember what happened after that. As he said:

- E** I cannot remember how I left the restaurant. I cannot remember whether I drove or somebody drove me (witness referred to evidence of SP14). I do not remember having gone to toilet during the lunch. I do not remember saying bye-bye to Ah Ming. I do not remember what time I left. I cannot remember whether it was daytime or night time. I remember what I was wearing in the morning. It was golf T-shirt and a pair of long black pants.

- F** In explaining what he could remember, he said:

- G** My next memory is waking up at some police station. I was handcuffed. Officers in plain clothes were holding me. They were around me. They wanted to take me somewhere. I think to the hospital. At that time, I did not know where they were taking me. I did not know in which police station I was in. I did not realize the clothes I was wearing. I cannot remember whether I was wearing my footwear. I was not aware whether I was wearing my own clothes. Subsequently, I know where I was taken. I was taken in a van. Subsequently, I arrived somewhere. I presume it was the hospital. I presume it was the hospital, as where else would they be taking me? At this place, I was shown to an Indian lady. She was somebody there. She wanted to take my blood sample. I did not know whether she was a doctor. I vaguely remember having seen SP8 on that night. This was the Indian lady I was referring to. I cannot remember what else she did.

- H** I remember being pricked by a needle. I cannot remember anything else in relation to that. I do not remember urinating for urine sample to be taken. I do not remember signing any consent form for blood and urine samples to be taken. I do not remember signing a consent letter at the police station. I do not remember having any conversation with the Indian lady. I remember the evidence given by SP8 on P17 (P17 dirujuk kepada saksi). This is my note. I can recognize it as my handwriting. It is a bit untidy as compared to my
- I**

normal handwriting. I remember writing the note. I also remember asking someone to contact my wife. I cannot remember who I asked. Prof Ernest Yeoh was in charge of the Accident and Emergency Unit of the Universiti Hospital in Petaling Jaya. At the time, I wrote the note I assumed I was in the Universiti Hospital and the doctor known as Prof Yeoh. As far I am concerned, Prof Yeoh was working at the Universiti Hospital on the day of the incident. I did not know that he had been transferred to the GH. Prof Yeoh is a friend of mine for 20 years. He had arranged for me to do certain medical tests previously at the Universiti Hospital. The telephone number on P17 is the telephone number of my wife. I wanted to contact her because as a responsible person I must tell her that I have been arrested so that she can help me. I contacted her as I am very close to her. We have no children. She would have been worried as I had not gone home. The 'Musa Hitam' in P17 is Tan Sri Musa Hitam, the former Deputy PM. He is a friend of mine. I remember shouting to some people in a room to call my wife. I don't know where the paper (P17) came from but it is my note. In the hospital, I do not remember whether the handcuff was on or not.

A

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There was a mass confusion in my mind when I got up in the building. People were shouting at me calling me a murderer. I cannot remember who. I do not know why they were calling me a murderer. I would think they were calling me a murderer as I was in handcuff and had some injuries. I did not even know I was arrested. I cannot remember being told for what I was arrested.

D

At the hospital, I did not feel physical pain. It was more mental confusion. I was drowsy. I was feeling sticky and sweaty. I did not have any other feelings in the hospital that I can remember distinctly (saksi dirujuk kepada P17):

E

Q: You appear sure that you were arrested at 5pm. Why did you write that?

A: I am not sure why I wrote that. I do not know why I put 5pm. I cannot remember whether the police took me somewhere else from the hospital. I was taken away by the police from the hospital. That I remember. I thought I was at the Universiti Hospital. I was taken back by the police in a van. I was taken back to a police station. I am not sure whether it was the same police station. When I arrived at the police station I was not aware of the clothes I was wearing. That night I slept in the police lockup. There were two others in the lockup. I slept in the lockup. I do not remember waking up. I remember being taken to a kind of court in the morning. I do not know the time I had a new set of clothes. They were my clothes. It was given to me by the police officers. I cannot remember changing into my clothes.

F

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I appeared in the court on the 23rd. It was before a Chinese lady. She was a Magistrate. I know the purpose why I was taken there. One of the police escorts told me that it was to take a remand order against me. The police escorts told me that a lady was fatally shot the previous night and that I was responsible. I still disbelieved it. Earlier it was shouted to me that I was a murderer.

H

After the court session I was taken back to the police station. By then, I knew which police station. They called it IPK. It is across the road from Pudu Jail.

Later in the day, the accused met his family members consisting of his parents, his wife, his sister Janet Lee, his uncle Thomas Lee and Mr KK Wong. As to what transpired at that meeting, he said:

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- A** I broke down when they said that the allegation was true. I broke down as we don't go around killing people. Apparently, I shot and killed someone. It was a terrible thing to have been done. Coming from my own family, it had to be true. I apologized to my father for the shame that I had brought. At that time, I already knew as I had already given my cautioned statement to ASP Thomas Woon. He had explained to me. Without being told, I did not know that I had shot someone. Until today, I never knew on my own that I had shot someone.
- B**

The accused then explained the state of his health. He has gastric problem and gets cramps in the legs often at night. One day in 1991 while having his shower, he experienced tightening of the chest, palpitations, difficulty in breathing, a tingling sensation at the finger tips, numbness of the hands and sweaty. He felt that his heartbeat was like bubbles being blown out of the water. The symptoms disappeared. He then went for dinner with his wife to a restaurant. There the symptoms reappeared. They went to the Pantai Medical Centre. His blood was tested and an ECG was done. It was found that he had low blood sugar. He was given a glass of glucose. He then recovered within ten minutes. The following morning, he had the same symptoms though not as severe. He saw Dato Dr Ridzwan Abu Bakar (DW2) at the Pantai Medical Centre. The accused then said:

- C**
- D**
- E** Dr Ridzwan did a series of tests even the treadmill. Also blood tests. I told him of the incident the night before. He assured me that there was no heart problem. But he confirmed that I had low blood sugar. He also gave me a glucose drink. I felt much better after that. He reminded that I take glucose with my drink Milo. He also asked me to carry some sweets just in case I need it. That morning before seeing Dr Ridzwan, I had quite a full breakfast as I missed dinner the night before.

- F** About a week later, the accused had the same symptoms while in his house at about 8pm. He met DW2 at the Pantai Medical Centre. The accused said:

This time, we managed to contact the same Dr Ridzwan. He met us at the hospital. He did the same tests. He confirmed that I was definitely suffering from low sugar in the blood. The reading was 3.4. I cannot remember.

- G** The accused said that since 1991, he has been taking glucose constantly with his drinks. He had also been treated by Dr Thiagi ('DW6'). As the accused said:

- H** I consulted Dr Thiagi as a friend. He said there is no medication. He told me to take lot of fluids. He said 100-plus is good. I have consulted Dr Thiagi on my complaint of low sugar. I play golf with him. He has taken my blood on many occasions. But I don't think specifically for sugar.

In 1999, Dr Ernest Yeoh of the University Hospital did some tests on the accused. As the accused said:

- I** They did a chest X-ray. There was nothing wrong. They did an ultra sound. The pancreas, liver and the kidneys were all normal. They also did a blood test. There was nothing alarming.

The accused had similar symptoms as he had experienced before in the Sungei Buloh prison. He has been allowed to keep a thermos flask for hot water to be mixed with glucose. He also testified on the use of his gun. In his cross-examination, the accused said that he has had the symptoms on about five occasions. The experience he had in 1991 while having a shower was the first. Prior to that, he did not have the symptoms. He described an episode he had in a golf club. The accused said:

A
B

The fourth episode was a few years later in 1994 or 1995. It was at the golf club. I thought it had something to do with my heart. This time it was not at 8 o'clock. I was driven in a buggy to the lounge. At the lounge, I lay down. We left about three hours later from the lounge. I was lying in the lounge. They said I was out for sometime. I could have dozed off or blacked out. My wife went to look for glucose. I took sweets in the buggy. I took 100-plus. I took the sweets before I arrived at the lounge. I took the 100-plus after I recovered. They woke me up. At that time, I was not feeling very hungry.

C

When asked what will happen to him when he gets drunk, the accused said:

In my younger days, I used to get drunk more. If one were to carry on drinking one will eventually get drunk. In my case when I am drunk I will sit down somewhere and doze off. If I am high on alcohol my breath will smell of alcohol.

D

...

If I have a hangover, I will feel generally very sick. That would be the situation when I drink a lot. When I get drunk, I quietly sleep off.

E

As he said further:

When I feel I am high or above the limit, I ask her to drive. By above the limit, I mean 80 ml and above. It is equivalent to four drinks.

F

With regard to his consumption of alcohol on the day of the incident, he said:

On 22 August 2000 by taking six glasses of beer, I believed I will not get drunk. My limit would be six glasses. Beer is a bulky drink. The alcohol content is less. Beer is called the long drink. I have not tried more than six glasses. If I have taken more than six glasses on that day, I cannot remember I cannot remember how many glasses I drank on that day. My normal is six glasses.

G

With regard to his loss of memory, the accused said:

It is not that. I do not want to remember purposely. It is not true that I purposely do not want to remember. It is also not true that I purposely do not want to recall shooting the lady.

H

The accused said that it is not true that he was highly intoxicated on that day. In commenting on exh P17 he said,

I wrote P17. In that letter I asked Prof Ernest Yeoh to contact my wife and Musa Hitam. Musa Hitam is my close friend. It did not cross my mind to call fellow directors or my parents.

I

- A** He said that he had no recollection as to the direction he was driving that day prior to the incident. In his re-examination, the accused said that he keeps a tin of glucose and 100-plus drinks in his office.

DW4 has been married to the accused for the past twenty years and they have a happy marriage. She said that the accused will normally go to office before lunch and return by 5pm. She added:

- B** If he was going to come back late, it is his habit to call me and inform me.

She testified on his drinking habits. As she said:

- C** As a matter of preference, he would normally drink whisky. It will be Black Label or Famous Grouse. He also takes brandy and beer. Beer is not a preferred drink of Kenny. He would normally have beer after playing golf. He would have three or four glasses after playing golf. Sometimes, he drinks more than three or four glasses. I have seen him having five glasses. I also play golf. Most of the time, I play golf with him. The other times, we play with our respective friends. He has about six to seven drinks of whisky. This is when he has his dinner. Sometimes, he exceeds that. He then takes eight to nine whiskies. I have seen him drinking a lot more on occasions during company dinners, birthdays and anniversaries. Then he will take easily three-quarter to a bottle of Black Label.

When he takes beyond the dinner drinks, he will be normally merry and jovial. He normally hugs his friends and family.

- E** She also gave evidence on the various occasions when the accused was not well and her visits with him to the Pantai Medical Centre to see DW2. She said:

- F** I began observing his chest pain, palpitation and sweating sometime in 1991. There was an occasion when I observed it. One night in October 1991, he was having his shower. He came out dripping wet telling me he did not feel good. He had his hand on his chest. He said he felt bubbles coming out from his chest. I told him to sit down and rest for a while. After about ten minutes, he went back to finish his bath. This was about 8pm. After getting dressed, he rested for about 20–30 minutes. Then we went for dinner. We would normally have tea at about 5pm. The tea will be with condensed milk and glucose and anything sweet. On that day, I cannot remember what kind of tea we had. But definitely we had tea. We went to the Sri Thai Restaurant opposite Petaling Jaya Hilton. As soon as we sat down, Kenny said he was not feeling good. He said his chest was tightening. He had nausea. He had numbness on the hands and sweaty palms. He did not look good. His face was very pale. He told me to call his mother, Datin. The mother is Datin Paduka Ruby Lee. She was at that time the Secretary General of the Red Crescent. He was worried he would get a heart attack. That was why I called his mother. She advised that I should get him to lie down in the restaurant. It was impossible to lie down in the restaurant. So we rushed home. I think I drove the car. When we got home, Kenny was still not feeling good. He thought he was going to get a heart attack. We did not have any food or drinks in Sri Thai. I then rushed him to Pantai A & E. I drove. There was an Indian male medical attendant. He did an ECG of Kenny and he did a blood sugar test. I was there all the time. The attendant assured me that Kenny was not having a heart attack. He said that Kenny's blood sugar level was very very low. The medical attendant gave a

A In describing the events that took place on the morning of 22 August 2000, she said:

On 22 August 2000, Kenny was supposed to have a Board meeting but he forgot. So I woke him up and he quickly got dressed and went to the office. The secretary called the house. Kenny finds it very difficult to get up. I have to physically wake him. That morning was no exception. He left the house at about 10.30am.

B

The next she heard from the accused was at about 7pm. As she said:

He called me about 7.15pm. It was just after 7pm. It was by phone. He told me that he was on his way home. He told me that he is OK and not to worry. He said that as he is normally home by 5pm. I am sure he knew I was worried and he was not back. There was no contact after that. He called the house. I think it must be mobile phone. I am sure it was mobile phone.

C

Some policemen came to her house between 8.30 and 8.45pm to check on her address. At about 9pm, she was informed by the police that the accused had shot somebody. She went to the police station at about 10.30pm. She could not meet the accused as:

D

ACP Hadi Ho told us that it is better to let Kenny sleep because he was in no condition for us to see him.

E

The following morning, she took a new set of clothes for the accused at the request of the police. She met the accused with some other family members at 2pm. In describing the meeting, she said:

We managed to see Kenny. Hadi Ho was with us with some other police officers. Then Kenny was brought in. It was at the conference room. Kenny broke down. When he walked in he was shocked and he broke down. We hugged him although he was in handcuffs. We told him not to worry. He said he was sorry and brought shame to the family. This was after the family told him that he had actually shot a lady. He just said he cannot remember anything and cannot recall what happened. ... After Kenny broke down, ie he cried, he said that he was sorry for bringing shame and embarrassment to the family. He said he could not recall anything. I did not ask him specifically about the phone call he made to me. I was with him for about 20 minutes.

F

G

She said that the accused is right handed and that he is always hungry even after a heavy lunch. She added:

By 3pm after lunch, he is hungry again. This happens consistently.

H

In cross-examination, she said that the accused does not snore at all. In summarizing the illness of the accused, she said:

As far as the palpitation, chest pain and sweating is concerned, he had four episodes. Three episodes in 1991 and once when he was playing golf in 1995. Other than that no more incidents.

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His feeling of hunger started in 1991. She said that they normally go to bed between 1–2am and the accused gets up about 1am. She said that she had

been told that the waking up problem of the accused is related to low blood sugar, perhaps by DW2. She was then asked: **A**

You came to know about the relationship between low blood sugar and waking up problems only recently after this case started.

She replied: **B**

Yes, before the case started.

She denied that her evidence on hypoglycaemia is to suit the defence of the accused. In her re-examination, she said that the first time she knew of the accused's low blood sugar was in 1991. Upon being asked about Dr Thiagi ('DW6'), she said: **C**

I heard of Dr Thiagi. He is one of my husband's doctors. I am not aware of my husband's visits to Dr Thiagi.

She was then re-examined about her knowledge that waking up late may have something to do with the accused's low blood sugar. The examination runs as follows: **D**

Q: When did you first know that waking up late may have something to do with your husband's low blood sugar?

A: I cannot remember. Now I know this for sure.

Q: Did anybody tell you this recently? **E**

A: I cannot remember. I am not sure whether this was told to me by Dr Ridzwan.

To a question by me to clarify her evidence on what they have for tea, she said:

I made a mistake here. Normally, he will have tea without glucose and some cakes. **F**

DW5 is currently the executive director of Green Mountain Holdings (M) Sdn Bhd. He has known the accused for about 20 years. He said that he had seen a tin of glucose on the table of the accused in the office. He had also seen him drinking 100-plus. He described the Board meeting that took place in his office in the morning of 22 August 2000 in the following words: **G**

It was the directors' meeting scheduled for 11 o'clock. The accused attended the meeting. The meeting started at 11 o'clock. The meeting was attended by I, the accused, Lim Thang Soon, Liew Yong Fee and William Cheong. We were discussing the future of the company and in relation to the China project. We were just talking about bringing back the money from China. It was not a problem. There was some delay in the money coming from China. There was one director who had problem of conflict of interest. But it was sorted out at that meeting. We talked about other things also. The meeting ended at about 1.30. There was no break in between. The director who had the conflict of interest was Mr Liew Yong Fee. **H**

DW5 said that at the meeting the accused was his usual self, that is to say, he will just listen and does not talk a lot at meetings. After the meeting, they **I**

A adjourned for lunch. He said that the accused ate some food and had some beer as well. He then added:

Mr Lim paid the bill for the food and few bottles of beer. He then left. That was about three o'clock. We were there till about 7pm. At 3pm, I, Kenny and William Cheong were left behind. Kenny and William paid as whenever they order additional beer.

B He cannot remember the number of glasses of beer that the accused drank. In describing the effect of the beer on the accused, DW5 said:

C All of us went to the toilet but not at the same time. I know that the accused went to toilet. I think it was three to four times. When he went to the toilet he got up and walked straight. He was not affected by the drinks. ... When the accused left the restaurant he looked okay. When Kenny left he was normal. At 7pm, Kenny's speech was as usual. Throughout the lunch till 7pm his behaviour was usual. He did not misbehave in any way at the lunch. At 7pm, we left together. I saw Kenny walking towards his car. That was the last I saw Kenny till I heard of the incident.

D In his cross-examination, he said:

The way we drank was slow. Kenny cracked jokes as we were drinking. Our mood was happy and jovial. While we were drinking slowly no food was taken. Other than jokes Kenny talked about golf and about his travel to Australia. ... While drinking the accused was talking more than usual.

E Upon being questioned about the tin of glucose in the room of the accused, PW5 said:

I have seen the glucose in his room at that time. I might have first seen in 1997 or 1998. I deny there was no glucose tin on accused's table.

F In his re-examination, he said that at the drinking session the accused and William talked more than usual.

G Harcharan Singh Tara ('DW8') is a Chemico-Legal and Forensic Consultant in private practice now and is involved, inter alia, in the examination and interpretation of alcohol intoxication cases. He retired as the Director General of the Chemistry Department, Malaysia. He holds a Master of Science degree from the Imperial College of Science and Technology, London. He is a Fellow of the Malaysian Institute of Chemistry, Associate of the Royal Institute of Chemistry (UK), Professional Associate Medallist in Chemistry of the Malaysian Institute of Chemistry and a former Registrar of the Malaysian Institute of Chemistry.

H He has attended numerous national and international conferences in forensic science. He has written many papers. They include papers entitled *Examination of Handwriting and Signatures* [1995] 3 MLJ i; *Expert Evidence in Civil and Criminal Cases* [1995] 2 MLJ vlv; *Alcohol Intoxication — How much is too much?* [1995] 3 MLJ xlv; *Some Observations of Alcohol Intoxication in Local Subjects — The Medical Journal of Malaya* Vol XXII 1965; *Urine Blood Alcohol Ratios in Local Subjects — the Medical Journal of Malaya* Vol XXVIII 1974.

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In relation to this case, the following facts were made available to him:

- (a) Mr Kenneth Lee's weight at time of incident was 74 kg, ie 162 lbs.
- (b) He had consumed presumably six glasses of Tiger beer. A small bottle of this consists of 325ml of beer of 5% concentration of ethyl alcohol (Ethanol).
- (c) Meal taken at about 2pm which lasted until 3pm and which consisted of a small bowl of rice, omelette, prawns and fish.
- (d) Passed urine about three to four times during the meal and drinks.
- (e) Started drinking at about 2pm and ended at about 7pm.
- (f) Incident took place at 8.05pm.
- (g) Blood sample taken by doctor at 1.20am and urine sample at 1.30am.
- (h) The blood alcohol concentration was 198mg per 100ml of blood and the urine alcohol concentration was 257mg for 100ml of urine.
- (i) He was a regular drinker.

DW8 was asked to determine the blood alcohol level of the accused at 8pm. By using the Widmark Formula, explained in his article as reported in [1995] 3 MLJ xli, he said that the maximum amount of beer that the accused would have had is about seven small bottles of beer of 5% strength. He said that when a person drinks alcohol he reaches a peak period of intoxication. This period is reached when the urine alcohol level is higher than the blood alcohol level. The urine alcohol level is then divided by the blood alcohol level. It is an established fact in toxicology that if the result of the division is between 1.28 and 1.33 then the alcohol is at its equilibrium peak level. Therefore, at any point of time prior to that the blood alcohol level will be on the ascending stage. This means that at that prior point of time the blood alcohol level will be lower. In this case, the urine alcohol level was higher than the blood alcohol level. A division of the figures produces a result of 1.29 and therefore falls within the equilibrium peak level. It follows that at any point of time prior to the taking of the samples from the accused his blood alcohol level will be lower. In explaining the concept of a peak period, he said:

- Q: Why did you make a finding that the accused had reached his peak when blood and urine samples were taken?
- A: The absorption of alcohol from the stomach is dependent on certain variables. One variable is the amount of food in the stomach; the amount of fluid in the body. The absorption from the stomach is usually very little and is slowed down by many factors. Two of the factors are what I had just mentioned. Most of the alcohol is absorbed from the duodenum from where it is carried around the body and is broken down by the liver (90% of it) into by-products. There is a phenomenon called gastric emptying. The valve between the stomach and the duodenum is called the pyloric valve. It is well known that when there is a lot of alcoholic fluid in the stomach especially when the drink is concentrated the pyloric valve closes up and does not release the alcohol for sometime. That may be another delaying factor of absorption by the duodenum into the blood stream.

- A** Q: What amount of fluids are necessary to create this situation?
 A: A strong alcoholic drink.
 Q: Can the expression 'strong alcoholic drink' refer to concentration as well as volume?
 A: If it is a strong drink the volume is less. If it is not a strong drink the volume has to be more to come to the same alcohol concentration.
- B** Q: Can six glasses of beer bring about a concentration that can be classified as concentration?
 A: It will depend on the spacing of the drinks. Therefore three of the factors that can contribute to the delaying of the process of absorption of alcohol into the blood, and thereby intoxication, are food in the stomach, amount of fluid in the stomach and the closing of the pyloric valve. Intoxication only takes place when the alcohol laden blood reaching the brain. For blood absorption to be delayed it could be just one of the factors.
- C**

In this case, the peak point was reached at about 1.20am. A urine blood alcohol ratio of 1.28 to 1.33 is attained when the alcohol is at its equilibrium peak level. If the alcohol level is still rising then the blood alcohol level will be greater than the volume alcohol level. Consequently, the ratio will be different from the equilibrium ratio. If the urine alcohol and the blood alcohol ration is much more than 1.33 then it will be the descending level of the concentration of alcohol in the body. The range of 1.28 to 1.33 represents the peak ends of blood alcohol and urine alcohol in the body. When the blood reaches the peak, it takes about half an hour to one hour for the urine to reach its peak. In the graph opposite p 4 the small 't' represents the time lag, usually half to one hour between the blood alcohol reaching its peak and the urine alcohol reaching its peak. This is the period that represents the 1.28 to 1.33. If blood is taken during the period of the two peaks it will be in the range of 1.28 to 1.33. This is an established fact in toxicology. The ratio in this case was 1.29. According to the chemist report the blood alcohol level was 198mg per 100ml of blood and the urine alcohol level as 257 mg per 100ml of urine. The calculation is done by 257 divided by 198. This equals 1.297. After the peak is reached, the alcohol in the urine level will be higher than the alcohol in the blood level.

- E** This ratio is an established scientific fact. (Witness referred to p 14 of D101) My conclusion is at p 17. (Witness asked to read first two para of p 14). If the samples are taken soon after consumption of drinks the absorption would still be taking place and the peak levels may not have been reached. (Witness referred to p 14) The fullest way to write the ratio is 1.33:1. The several graph opposite p 4 shows the blood alcohol level against time. It would look a jagged saw tooth going upwards. Subsequently it would rise slowly and fall down. I have put the peak between 12 and 1am. My conclusion is that at about 8pm the alcohol blood concentration in the accused would have been in the range of about 92–119mg. I have stated the symptoms in the report.
- F**
- G**
- H**

DW8 then calculated the blood alcohol level of the accused at 8pm. As he said in exh D101:

- I** The excretion rate of alcohol in our particular case, I will take a range, ie 15mgs to 20mgs per hour, providing there are no other intervening or interfering substances like for example food or fatty substances in the stomach or some other phenomena which will have the effect of delaying the absorption of alcohol into the blood stream. Taking 5.3 hours and 15mgs excretion rate

- A • It should be noted that the categories as stated overlap, hence the signs/symptoms defining these categories overlap also. The signs/symptoms in all categories may significantly impair driving regardless of their relative degree of severity or detectability.

SOURCE : Kurt M Dubowski PhD Professor of Biochemistry and Toxicology, Medical Centre, University of Oklahoma City, Oklahoma.

- B In conclusion, DW8 said:

From the above calculations and research reference it could be summarized that the alcohol intoxication level for Kenneth Lee could be categorized as mild to moderate at the time of incident, ie 8pm.

- C With regard to the preservation of blood and urine samples, he said:

For purposes of measuring the alcohol concentration in the blood usually sodium fluoride is added as a preservative to inhibit the ethanol metabolism by micro organism. Before doing a test, I ensure that a blood sample has not degenerated by doing a visual test which will show if there is a gas formation or whether there is smell. If there is smell it means the sample is deteriorating. Normally we do a culture for post-mortem samples. Even in other cases a culture test can be done. This can be done to make sure if there is smell. If there are microorganism the level of alcohol can be increased or decreased. If we are not sure a culture test can be done.

- E In the case of urine, it is not necessary to add any preservative. It can be kept in the refrigerator. But still sodium fluoride can be added to ensure that there is no deterioration.

In his cross-examination, he said that the time taken for a glass of beer to be absorbed will depend on whether there is food in the stomach. If the food is fatty the absorption is delayed. When he was referred to the food that the accused had consumed, he said:

- F If all that food is in the stomach the absorption will be even faster.

He was then referred to p 17 of a book entitled *Drink, Drugs and Driving*. In commenting on it, he said:

- G I do not agree with the author when he says that the additional effect of food is relatively much less important in the case of beer. As long as there is some food in the stomach there will be delay of 1–1½ hours depending on the amount and type of food.

On the ability of a person to drive when he is in the level of intoxication as the accused, DW8 said:

- H It is very subjective to say whether a person in this category can drive a car. Now I say that a person in the second category can drive a car. Sometimes there is increased self-confidence in this category. A person in this category can drive 12 km. He will drive consciously without being in an automated state. Automated driving in alcohol intoxication is only when the alcohol level is very high. Automated driving due to alcohol does not come under the second category.

- I With regard to the signs and symptoms at the different levels of intoxication, he said:

The level of decreased inhibition varies from person to person. Some people have been known to be very quiet after a drink. Some are otherwise. I have not come across a case where a person is normal and then explodes. (Witness referred to p 35) The accused falls under the second category. This guideline has been generally accepted but there may be cases that do not fall within it.

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He said that it is possible for an intoxicated person to urinate and defecate in his pants. He added that even a non-intoxicated person can do that. DW2 was questioned on the difficulty of fixing the blood alcohol level of the accused at 8pm in the following way:

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Q: We have the evidence of Prof Marks who said that it is very difficult to determine the alcohol level in this case at 8.00 p.m given the 198 mg at 1.30am.

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A: I do not agree with it because one can calculate within a range. I do not agree with the learned Professor.

In his re-examination, DW8 said that his calculation of the blood alcohol level of the accused at 8pm would have taken into consideration the relevant variables. He also said:

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One must have enough knowledge and experience to be able to do the calculation to determine the level of intoxication at a prior point of time. This is within my field of expertise as a chemist. I agree that it is dangerous and not wise to determine a specific figure at any given prior time.

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The accused called several other witnesses to support his defence of automatism. They are Dato Dr Ridzwan Abu Bakar ('DW2'), Professor Dr Vincent Marks ('DW3'), Dr Thiagi Selvanayagam ('DW6'), Dr Elizabeth Ann Rajan ('DW7') and Tan Sri Dato Seri Dr Mahadevan ('DW9'). DW2 and DW6 had treated the accused prior to the incident while the other witnesses expressed their opinion on the medical condition of the accused at the time of the incident.

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DW2 is a cardiologist at the Pantai Medical Centre, Kuala Lumpur. He first saw the accused on 23 October 1991. From tests that he conducted, he found that the accused did not have a heart disease but had a low fasting blood sugar of 71ml which is 3.9 millimoles per litre. He then said,

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When he came to see me, his fear was whether he had heart disease. I told him he had no such disease. With regard to what happened to the accused on the 22nd, I was not able to know what he did in relation to the symptoms. I have no recollection of him telling me that he had been to the A & E. I have no recollection as to whether he told me that an Indian HA or hospital staff at the A & E gave him glucose after testing him.

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DW2 saw the accused again on 30 October 1991. A random blood sugar test showed that his blood sugar was 3.9 millimoles per litre. The ECG test was normal. In elaborating on his findings, DW2 said:

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In view of the low blood sugar, he was given oral glucose by the hospital staff under my advice. It was done, firstly, because of the low blood sugar. Secondly, the symptoms were suggestive of low blood sugar. Thirdly, it is an

A important test to determine the diagnosis of low blood sugar. Low blood sugar is hypoglycaemia. There is a special significance in the reading of 3.9 mm on that day. It means that the patient has hypoglycaemia. I would expect a patient who has eaten to have a blood sugar level of higher than that. If he had eaten up to four hours before the blood sugar should be in excess of 4.0. I made a provisional diagnosis of hypoglycaemia. The first test done was taken into consideration for this conclusion.

B On the second occasion, I was impressed by the dramatic recovery he made following the taking of oral glucose. I advised him, like a good doctor, to give up smoking and drinking. I added that should he choose to continue to drink, he had to do it on a full stomach. The other advice I gave was that he ought to be investigated further for the cause of the hypoglycaemia. There are over 100 causes of hypoglycaemia. The only medicine I prescribed on that visit is novalucid. This is an antacid prescribed for heart burn. In relation to hypoglycaemia there was no prescription but just advice. I asked him to take frequent meals and to carry sweets or glucose with him. I did tell him to keep up his blood sugar by taking sugar frequently.

C On 9 February 1996, DW2 saw the accused for the third time. On the previous day, the accused had experienced two episodes of sharp left sided chest pain while playing golf. DW2 conducted a treadmill stress test on that occasion. With regard to the results of his tests, DW2 said:

D I conducted a treadmill stress test on that occasion. The result was similar to the first test conducted on his first visit. He brought some blood test results on his own. He was not referred to me by a doctor. A whole list of blood tests were done by the Pathlab. There is nothing in relation to hypoglycaemia. The liver function test was done and it was normal.

E The accused saw DW2 for the fourth time on 10 February 2000. In describing this visit, DW2 said:

F He came specifically requesting for a treadmill stress test. I do not know why. He just wanted the test to be done. The result was similar to the other two occasions.

G There was no other visit to DW2 by the accused. DW2 said that the complaints of the accused are common to heart problem and hypoglycaemia. In cross-examination, DW2 said that on both the occasions he did blood tests on the accused and made a diagnosis of provisional hypoglycaemia. When asked to describe the symptoms in a hypoglycaemia patient, he said:

H In the case of person undergoing a hypoglycaemia attack from mild to severe it can happen gradually or abruptly. It depends on the underlying cause.

I A hypoglycaemia patient can be confused and aggressive. In the mild stage, the patient will be conscious. That is when he will have the symptoms. The symptoms will be related to the degree of hypoglycaemia. The early symptoms will include trembling, sweating, palpitation, lethargy, drowsiness, chest pains. The next stage will be (pallor), ie (paleness), confusion, irritability. At this stage he will be conscious. There will be hunger at this stage as the body system is trying to supply the sugar. At the mild stage walking will be normal.

A He also said:

The complaint made by the accused's wife was that he had faintage, sweating and palpitation,

B He then said that it was established in 1991 that the accused had hypoglycaemia. When the accused saw DW6 on 1 October 1991, DW6 had advised him to see a cardiologist. At that time, he did not pinpoint the accused's complaint as one of hypoglycaemia. When he said this, he was asked:

Why then did you advise Kenny to have a sweet drink?

C DW6 replied:

Although I did not pinpoint the cause to be hypoglycaemia I suggested sweet drink before being brought in.

He was then asked:

What if he was not suffering from hypoglycaemia?

D DW6 replied:

As a first aid, a sweet drink is always recommended. Secondly, Kenny was not a known diabetic. To be hypoglycaemic, one must be a diabetic.

E He was then referred to the patient card of the accused ('exh D91') in relation to the entry for 12 November 1998. In explaining the entry, he said:

On 12 November 1998 when I wrote hypoglycaemia leading to syncope on D91 I was referring to the finding made earlier by Dr Ridzwan.

When questioned by me as to why that entry is below the last line, he said:

F The new page may not have been attached by my staff as yet or I could have retrieved the card myself and scribbled as Kenny was rushing to hospital.

With regard to entries made in a patient's card, he had said earlier:

G I agree that the record made in a patient's card is important. Any examination done must be recorded in the card. If there is something of importance we would record it. Sometimes we record even after a few days. Sometimes I fill in particulars for a patient even up to three days later. This post-recording can happen once in six months. In this case, I did the record in a hurry as I saw accused in a hurry. When I do a post-recording, I date it back to the date of examination.

H He said that he did not know that DW2 had diagnosed the accused as a case of provisional low blood. Upon his re-examination on the matter of making entries in his patient's card, he said:

I There are times when I have made notations on a patient's card not immediately. This is rare. This arises if patient is not in front of me, for example, a phone call or if I had seen the patient on a housecall when the card is not with me. If I had seen a patient in a hurry. ... It may also be when my staff were not around. Normally, I write the notes when the patient is in front of me. My staff attach the last card to the notes. It does happen that the card is filled up and the next empty card is not added. (Witness referred to D91A)

(Entry for 12 November 98 referred to) If there was no new card I would have done as in this case. Unless the case notes are going to be longer. I am sure I can find some occasions when I have done this.

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DW3 was formerly the Dean of Medicine and currently Professor Emeritus of Clinical Biochemistry at the University of Surrey. He was a Consultant in Chemical Pathology in the National Health Service. He is a Doctor of Medicine of the University of Oxford, a Fellow of the Royal College of Physicians of London and of Edinburgh and a Fellow and former vice-president of The Royal College of Pathologists. He is a member of the British Academy of Experts. He has had extensive experience in all aspects of clinical medicine especially its laboratory aspects. He has conducted research on all features of carbohydrate metabolism — especially hypoglycaemia — and is the senior author of a book entitled *Hypoglycaemia*. In addition he has published over 200 papers in the scientific literature on various aspects of insulin and glucose metabolism in relation to diabetes and hypoglycaemia. He has also made a special study of the metabolism of alcohol in man and has published extensively on the subject. He has acted as an expert witness, for both the prosecution and the defence, in the United Kingdom and abroad in cases in which hypoglycaemia and/or alcohol were key considerations. In this case he has been asked to express an opinion on:

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- (a) whether the events as recorded and described could have happened whilst the accused was suffering from hypoglycaemia, and
- (b) whether the accused's amnesia for the events is credible.

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To enable him to arrive at a conclusion he was furnished with various documents. They are the chemist report ('exh P48'), the autopsy report ('exh P72'), certificate dated 26 September 2000 by Dr Thiagi Selvanayagam ('exh IDD83'), letter dated 16 April 2001 accompanied by photocopies of medical notes of Dato Dr Ridzwan Abu Bakar ('exh D85'), witness statement from Tan Ah Chai dated 11 September 2001 ('exh D86'), witness statement of Dato Dr Ridzwan Abu Bakar ('exh D87'), witness statement of Dr Thiagi Selvanayagam ('exh D88'), notes of meeting with Dr M Mahadevan on 13 July 2001 ('exh D89'), witness statement of Noor Salasawati ('exh D90'), medical notes of Dr Thiagi Selvanayagam ('exh D91'), notes of proceedings as recorded by counsel with translation ('exh D92'), witness statement of Ng Kiew Ming ('exh D93'), newspaper cutting ('exh D94') and statement of the accused ('exh D95'). As many of the documents involved have not been properly tendered, I informed parties that their evidentiary value, though marked as exhibits, are subject to proper proof and relevancy. In a later part of his examination when referred to exh D92, DW3 said:

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I have not read every line as thoroughly as would be required to speak authoritatively on them. I have looked at the trial transcripts in particular reference to the parts which I considered to be relevant. I believe I have captured these relevant parts in my report. I have read the testimony of the accused.

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- A** In light of the fact that the hub of the defence advanced is one of hypoglycaemia and in respect of which DW3 had been asked to express an opinion, it is necessary to understand what it means. In order to do so, I consider it necessary to reproduce certain parts of exh D84.

- B** The blood contains a small but very important amount of a sugar known as glucose. The total amount of glucose present in the body at any one time is seldom less than about five grammes even after many days without food nor, except in patients with the illness known as diabetes, is it ever more than about 20g even after a very large carbohydrate (sugar and starch) containing meal. The amount, often referred to as the concentration, of glucose in the blood is ordinarily controlled within very
- C** narrow limits. The concentration rarely rising above 10mmol/L (equivalent to about 1.8g glucose per litre of blood) even after a large meal: nor does it fall below about four mmol/L (equivalent to 0.72g/L of blood) even after several days without food. A blood glucose concentration greater than 10mmol/L (corresponding to a blood glucose concentration of 180mg/
- D** 100ml) is termed hyperglycaemia; a concentration of below about 3.5mmol/L as hypoglycaemia but these terms are arbitrary and do not define a clinical condition. The remarkable control of blood glucose by the healthy body is only made possible by its ability to dispose of glucose into the tissues as fast as it is absorbed from the intestine during and immediately after a meal, and to put glucose back into the blood from its stores in the
- E** liver as soon as all of the food has been absorbed. This remarkable feat is called homeostasis and is analogous to the way that a thermostat maintains a refrigerator's temperature within narrow limits. The brain is the only organ of the body that relies entirely upon glucose as its source of energy. The liver, on the other hand, is the only organ that can liberate glucose into the circulation during fasting — and it does so at a rate just sufficient to
- F** supply the brain with its quota of fuel. The brain, because it has no stores of glucose itself, must rely upon a steady stream of glucose coming to it in the blood in exactly the same way as it depends upon a steady supply of oxygen. But whereas the dire effects of suffocation (lack of oxygen) upon brain function was well known those due to lack of glucose are much less
- G** well known. Evidence of brain malfunction begins to appear if, for any reason, the blood glucose concentration falls below a critical level, which is usually about 3.5mmol/L but can be higher or lower. Exactly what form this malfunction takes varies enormously both from individual to individual and in the same individual from time to time depending upon circumstances. Because the other tissues of the body such as the heart, kidneys and muscles
- H** can use fats from the blood as their source of energy they are not affected in the same way as the brain by a lack of glucose in the blood and they continue to function normally. One of the earliest actions of the brain when its glucose supply drops below its critical level is to activate the release of what are known as counter-regulatory hormones whose function it is to try and restore its glucose supply. The two most important of these counter-
- I** regulatory hormones are glucagon, which is made in special cells in the pancreas and adrenaline. Both these substances cause the blood glucose level to rise by stimulating the breakdown of glycogen, the storage form of

glucose in the liver, into glucose that is liberated into the blood. If for any reason the liver does not have any glycogen stores these counter-regulatory hormones cannot work — or do so only very poorly — and the blood glucose level continues to fall and both hypoglycaemia and brain malfunction become more severe. There are a large number of diseases in which glucose homeostasis or control of blood glucose is disturbed. Of these diabetes is both numerically and clinically the most important. It is characterized by a high blood sugar level that does not ordinarily affect brain function adversely. Treatment of diabetes is with drugs that cause the blood glucose level to fall sometimes to such low levels that brain function is impaired. This is the reason why hypoglycaemia is so closely associated in people's minds with diabetes — not because diabetes itself causes hypoglycaemia. There are about 100 illnesses that can cause hypoglycaemia. In about 6–10% of patients suffering from hypoglycaemia, no cause can be identified. Hypoglycaemia is an arbitrarily defined condition. Its importance stems from the fact that it can give rise to a wide variety of symptoms all attributable to a lack of glucose for metabolism within the nerve cells of the brain. It was for this reason that DW3 coined the term neuroglycopenia to describe them since they can only be alleviated by glucose. The earliest changes to occur as the blood glucose level falls in a healthy subject as a result of experimental intervention, or in a patient as a result of illness, are subtle changes in cognitive brain function and of which the patient is usually completely unaware. These are followed, as the blood glucose level continues to fall, by the release of what are called counter-regulatory hormones. The two most important of these are glucagon and adrenaline. The secretion of glucagon into the circulation cannot be perceived by the human brain but release of adrenaline, on the other hand, produces symptoms of which the subject is usually all too aware. These symptoms may include profuse sweating, a rapid heartbeat, tremor of the fingers, great anxiety, strange feelings in and around the lips and other sensitive parts of the body (paraesthesia), and a feeling of general unwellness. This condition, which DW3 describes as acute neuroglycopenia, is the one typically produced by the injection of insulin and is familiar to every clinician dealing with diabetic patients. If the fall in blood glucose level stops before it reaches a level of three mmol/L or less, either as a result of eating some food or in response to the counter-regulatory hormones themselves, the patient may never (consciously) know that they were in danger of becoming overtly incapacitated. They may, if they have been taught to recognize these premonitory symptoms of impending severe brain malfunction, abort its progression by eating or drinking something sugary. Sometimes, however, especially when hypoglycaemia is the result of illness rather than the injection of insulin, hypoglycaemia fails to produce these premonitory symptoms and the patients remain blissfully unaware that anything is wrong with them until their brain function has deteriorated to such a level that they are unable consciously to do anything about it. Whilst in this state of being neither comatose nor properly conscious a person may behave in a most uncharacteristic manner. This state is often referred to as hypoglycaemic

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- A** automatism during which the patient may perform quite complicated tasks such as driving a motor vehicle or holding an apparently intelligent conversation. When they subsequently recover from their hypoglycaemia they may, however, have absolutely no recollection of what they did during this time — it is as if they had been ‘sleep walking’. Such patients are suffering from sub-acute neuroglycopenia — to distinguish it from the more florid symptoms of acute neuroglycopenia — but what is now better known as hypoglycaemia unawareness. Whilst most patients in this state are quiet and often sleepy — but still capable of doing habitual tasks poorly — a small and unpredictable minority become aggressive and even violent for no apparent reason. Eventually they, like those with the more typical
- C** symptoms of somnolence, either lapse into coma or recover spontaneously or after being given a sugary drink or sweet. A patient whilst suffering from sub-acute neuroglycopenia may appear, to an independent observer, to be drunk or under the influence of drugs. This is often the first diagnosis to be given to someone who subsequently turns out to have an insulin secreting tumour or other illness capable of causing hypoglycaemia. The onset of
- D** convulsions or epileptic form fits as a result of hypoglycaemia is common in infants and young children in whom it may occur as many as in 70% of cases or more. It is also a well recognized and important late complication of hypoglycaemia in adults who may never have experienced a fit before. It would be rare for an adult to have a hypoglycaemic fit unless their blood glucose level had fallen below about 2.2mmol/L. Classically, patients whilst
- E** suffering from hypoglycaemia have no idea of the passage of time and if ‘brought round’ and their brain function restored to normal by giving them glucose to drink or by intravenous injection are bewildered by their surroundings. They have no idea where they are, what time it is or what has happened to them since they began to go hypoglycaemic. Hypoglycaemia
- F** used to be thought to of as rare — except in diabetic patients undergoing treatment with insulin. It can, however, develop in many illnesses — sometimes as the predominant or only feature of it and was so in 1% of all the patients admitted to the internal medicine wards of a teaching hospital in Turkey as recently reported. The subjective symptoms produced by hypoglycaemia may be comparatively mild to such an extent that the
- G** patients may themselves be quite unaware that they are impaired by it. This can go on for many years — 5–15 years is far from uncommon — before a definitive pathological diagnosis is eventually made. Patients with episodic hypoglycaemia often seek medical help only because others close to them, such as their family or work mates, notice their odd behaviour. Occasionally
- H** they note that these episodes are brought to an end by eating but this is far from universal. During their periods of hypoglycaemia, automatism patients may still appear to function — though less efficiently and skillfully than usual — but they themselves may have no memory of what they did. The characteristically dense and impenetrable amnesia for events that occur during periods of hypoglycaemia automatism can of course occur in other
- I** diseases of brain function such as epilepsy. It is only by obtaining an independent history from a by-stander of what, if anything, actually happened during the time the patient was neuroglycopenic that his medical

attendants can get at the truth. The patient either has no memory of the events or confabulates, ie makes up a story to explain the condition in which they find themselves when they eventually do 'come round' as their blood glucose level rises. Because of their unreliability as witnesses, DW3 has always advised, in any case in which (spontaneous) hypoglycaemia is suspected as the cause of a patient's illness, that a history be sought, by interview, from the spouse or close relative. Neuroglycopenic (autonomic) symptoms which are often referred to by laymen, and doctors alike, simply as 'hypoglycaemia' can invariably, and very rapidly, be reversed by raising the blood glucose concentration into the normal range, ie 4–10mmol/L. But whereas patients with diabetes who are being treated with insulin are taught early in the course of their illness to recognize the early premonitory symptoms produced by the release of adrenaline and so prevent deterioration into coma by eating or drinking something sugary others whose hypoglycaemia is secondary to illness rather than treatment seldom recognize its onset. Hypoglycaemia is almost always ephemeral: that is it lasts for a few minutes to a few hours at most because it either recovers spontaneously (or as a result of being given food) or, more rarely, progresses into coma. The only exception, and this is exceedingly rare, is when hypoglycaemia develops very slowly in elderly people who have an insulin secreting tumour of the pancreas. In them, and them alone, the blood glucose concentration is low whenever it is measured. Their symptoms are those of dementia or other chronic mental diseases and, in contrast to people with episodic hypoglycaemia; do not get better when their blood glucose concentration is raised by giving them glucose. Rarely, they may recover their full mental faculties if the tumour causing their hypoglycaemia is removed but usually they do not. Ordinarily therefore hypoglycaemia — and the symptom to which it gives rise — occurs episodically. There are often long periods — sometimes as long as several years — between 'attacks' though why this should be is still poorly understood. In between times, the patient may appear completely normal although intensive testing with batteries of appropriate laboratory tests will often — though not invariably — reveal the cause of their hypoglycaemia. Amongst the spectrum of signs and symptoms produced by hypoglycaemia is a behaviour pattern that may closely resemble that produced by alcoholic intoxication. Indeed many of DW3's own patients over the years were originally diagnosed as suffering from alcoholic intoxication before the true cause of their symptoms was discovered by measuring the blood glucose concentration whilst they were actually suffering from symptoms. The tell-tale difference between alcoholic intoxication and hypoglycaemia mimicking it, is that in the latter the blood glucose concentration is low during an attack and the abnormal behaviour attributed to it is abolished by intravenous glucose. In other words they can 'recover' remarkably quickly. In alcoholic intoxication, on the other hand, the blood glucose level is normal or high, there is an excess of alcohol in the blood, glucose has no effect upon the patient's behaviour and they 'recover' only very slowly as the body digests the alcohol in it by metabolism. A common characteristic of hypoglycaemia, whether produced naturally by disease or artificially as part of a scientific experiment, is the

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- A** onset of ‘irritability’ leading, in some cases, to anger that may be directed against themselves. Teasdale has recently described just such a case but every diabetologist will have seen it many times in his own patients. This state of mind has been designated haemodementia. A small but important minority of patients develop completely uncharacteristic aggressive behaviour that abates dramatically once their blood glucose level is restored to normal. This can be achieved by getting the patient to drink a sugary solution, eat something starchy or, in the most severe cases, by forcibly injecting them with glucose or glucagon. Once restored to normal the patient has no recollection of their deviant behaviour or of the passage of time during which they were suffering from hypoglycaemia. There is no evidence that aggressive behaviour occurs only in patients who display aggressive behaviour when their blood glucose level is normal: indeed in most cases in which it occurs such behaviour is totally alien to them and is consequently hard for their loved ones who have not witnessed it — and the patients themselves — to believe. Because hypoglycaemia can only be diagnosed with certainty by measuring the blood glucose level whilst the patient is actually suffering from its effects — and these are virtually always short lived — it is almost impossible to dismiss the possibility that a person’s aberrant behaviour was due to hypoglycaemia once it has been suggested that they were. Pointers to the probability that a person’s abnormal behaviour was due to hypoglycaemia whether it was as trivial as use of abusive language or ‘shop lifting’ or as serious as driving dangerously or behaving downright violently are:
- E**
- (1) apparently motiveless and totally uncharacteristic behaviour;
 - (2) a prior diagnosis of hypoglycaemia;
 - (3) subsequent diagnosis of a disease known to cause hypoglycaemia;
- F**
- (4) a rapid and complete change in the patients demeanour after being given something sugary to drink or eat.

Whilst none of these pointers is diagnostic in its own right they are collectively the best that can be done — in the absence of a blood glucose measurement made at the relevant time and which is exceptionally rare — to support a tentative diagnosis of hypoglycaemic automatism.

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The opinion given by DW3 is in the following terms:

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There seems to be very little doubt that during the evening of 22 August 2000 Kenneth Lee acting alone and in a most uncharacteristic manner killed Mrs Linda Lee and assaulted many other people with gun-fire in an apparently motiveless attack. Moreover, it is evident that during, but most probably in the aftermath, of the attack Kenneth Lee became incontinent of both urine and faeces. The cause of Kenneth Lee’s apparent ‘brainstorm’ is uncertain. Because no blood glucose measurements were made on Kenneth Lee at the relevant time, it is impossible to be certain that Kenneth Lee was suffering from hypoglycaemia at the time of the attack. It is likely however that he had a convulsion. His medical history is certainly suggestive of episodic hypoglycaemic attacks of undetermined origin. Although many of the causes listed in table one can be eliminated from further consideration too many remain for discussion of each one of them in detail to be fruitful. I do however

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attach particular significance to three sentences in Noor Salaswati's evidence. She says *'Kenny also has a problem with waking up in the morning. He usually wakes up about 10am to 11am in the morning. I would make three to four attempts to wake him up. I had to physically shake him before he would wake up. Even then he would often fall back on the bed to continue sleeping.'* This description fits well with that given by Ramaya s/o Muniandy who says *'He also did not wake up in the mornings'* and who replied to questions that *'I always had to wake him up physically'*. Difficulty with waking is always a strong pointer towards an 'organic' cause of a patient's hypoglycaemia as is the fact that it is precipitated by exercise such as golf. The possibility that Kenneth Lee has an abnormality of insulin secretion as the cause of his hypoglycaemia is suggested by the fact that he displayed many of the commonest features of this illness over the 10 years preceding the attack. Some 20% of the 59 patients with hypoglycaemia due to insulinoma seen at the Cleveland Clinic in Ohio had had symptoms for more than five years before the diagnosis was established. In some patients diagnosis was delayed as long as 30 years in keeping with the experience of others including myself. The authors (Dizon AM et al. Neuroglycopenia and other symptoms in patients with insulinomas. Am J Med 1999; 106: 307-10) of this report go on to say *'all patients presented with neuroglycopenic symptoms. Common neuro-glycopenic symptoms were confusion and personality changes or bizarre behaviour. About 40% of patients had amnesia for some hypoglycaemic events. The most common autonomic symptoms were diaphoresis (sweating) and tremulousness'*. No less than 27% of their patients had suffered hypoglycaemic convulsions and 39% had been given a diagnosis of epilepsy prior to establishing the true cause of their illness. 8% had been diagnosed as intoxicated by alcohol and 2% (one patient) as having had a heart attack. Other causes of hypoglycaemia can, however, closely mimic insulinoma and differentiation requires thorough laboratory investigation. I would be reluctant, in the absence of such investigations, to speculate on the ultimate pathology (cause) of Kenneth Lee's hypoglycaemic episodes such as that witnessed by Dr Ridzwan Bakar. This was clearly a less severe and dramatic episode than is postulated to have occurred on the evening of the 22nd August 2000 and to have had such tragic consequences. Incontinence of faeces does not ordinarily occur as a result of uncomplicated hypoglycaemia but is a recognized complication of hypoglycaemia-induced convulsions — though not one I have experienced personally. I have seen nothing in the evidence that excludes this possible explanation of an otherwise uncommon event. Faecal incontinence can occur as a result of alcoholic intoxication but only, as Dubowski points out, when the subject is almost in extremis from alcohol poisoning. Kenneth Lee's behaviour on the evening of 22nd August 2000 is consistent with him having entered into a state of hypoglycaemic automatism shortly after leaving the company of his fellow directors and getting into his car to drive home. His subsequent behaviour especially his faecal incontinence point strongly to him having had a convulsion from which he only recovered whilst at the police station and before being seen by a doctor. The cause of Kenneth Lee's episodic hypoglycaemia is uncertain and impossible on the evidence I have seen to attribute to any of the many causes mentioned in the introduction to this statement. The investigations necessary to establish the cause were not undertaken at the time of the original diagnosis and speculation at this time would be unrewarding. In the absence of a blood glucose measurement, there is no direct evidence that Kenneth Lee was suffering from hypoglycaemia at the time of the assault. It would, however, explain behaviour that was out of character for which there is no other readily

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- A** available medical explanation. It is perfectly feasible that hypoglycaemia and a hypoglycaemic convulsion could account for everything that happened to Mr Kenneth Lee between about 7.30pm when he left the company of his co-directors and when he recovered awareness in the prison some time later. The history of Kenneth Lee's alcohol drinking on the day of the offence is not compatible with the blood (and urine) analytical results. These, if accepted as correct, are difficult — though not absolutely impossible — to reconcile with the evidence.
- B**

In making a summary of his opinion, DW3 said:

- C** The evidence is not inconsistent with the suggestion that Mr Kenneth Lee was suffering from neuroglycopenia (hypoglycaemia) immediately prior to, during, and for a short time after his lethal attack on Madam Lee Good Yew (Madam Lee). It is not established beyond doubt that he was suffering from hypoglycaemia since no blood glucose measurement was made at the relevant time. Mr Kenneth Lee's amnesia for the events is both credible and supportive of the proposition that he was suffering from hypoglycaemia (neuroglycopenia) at the time of the offence. I have seen nothing in the evidence that is inconsistent with the above statements or with those of the various witnesses to the events of the evening. Alcohol was undoubtedly present in Kenneth Lee's body and may have compounded the effect of hypoglycaemia on his brain function but is unlikely by itself to be the complete, or sole explanation for the events that took place.
- D**

- E** In commenting on the consumption by the accused of about six glasses of beer each containing 330ml Tiger beer of 5% v/v alcoholic strength, DW3 said:

- F** Thus, Kenneth Lee drank about two litres of beer containing 40g of alcohol per litre. (alcohol weighs 0.8g per 1ml) or 80g in all. This is not an excessive amount for someone accustomed to drinking something in the region of 100–150g of alcohol a day. Most normal healthy individuals destroy alcohol in their body by metabolism at a rate of about 7–10g per hour. If Kenneth Lee did this, he would have had no more than about 20–40g of alcohol in his body at the time of the assault at about 8pm and would have given him a blood alcohol level of about 40–80mg/100ml. In the event, I understand no blood was collected for either an alcohol or glucose determination until about 1 am the following morning when samples of both venous blood and urine were collected. These were sent for analysis. Blood glucose was not measured. The result of the analysis for alcohol were reported as 198mg of alcohol per 100ml of blood and 257mg per 100ml of urine. His ratio of alcohol in urine to blood is consistent with both specimens having come from the same individual but difficult, though not impossible to reconcile with the history of alcohol consumption described above. There are several possible explanations for this: (1) unreliability of the witness(es) stating how much alcohol Kenneth Lee drank that day; (2) the specimens analysed were from someone other than Kenneth Lee but were confused with them; (3) unreliability of the assay or contamination; (4) Kenneth Lee did not, for some reason break down alcohol by metabolism at the normal rate of 7–10g an hour but a much slower rate of 1–2g an hour (a blood alcohol concentration of 198 mg/100 ml in a man weighing 74kg means that there is about 105g of alcohol in the body as a whole. This approximates the total amount of alcohol in the Tiger Beer he described as having drunk.) I am unable to comment on possibilities 1–3. I have, however, personally seen and investigated a man who metabolized
- G**
- H**
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alcohol only very slowly. Consequently, his blood alcohol level was almost the same some six hours after, as it was one hour after drinking. Some drugs and certain illnesses are known to reduce the rate of destruction of alcohol by metabolism in the body. Because I do not know the effect of hypoglycaemia on the rate of alcohol metabolism and am unaware of anyone who has undertaken research on this subject it would be inappropriate for me to speculate on what Kenneth Lee's blood alcohol level might have been at any other time than when it was measured. It is however known that some sugars, particularly fructose (Merry J Marks V effect on performance of reducing blood alcohol with fructose. *Lancet* 1967: 2.1328), can increase the rate at which alcohol is destroyed in the body.

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Upon being asked whether alcohol would have caused the accused to behave the way he did, DW3 replied:

C

If the element of hypoglycaemia is removed leaving only the element of alcohol presence in the body, I would find it very hard to accept alcohol as the sole cause on the evidence made available to me. The amount of alcohol consumed, that is, two litres of beer providing 80g of alcohol is a very common occurrence and in someone used to drinking that amount of alcohol would be very unlikely to produce effects we have heard about.

D

He was then asked whether his opinion would be the same on the assumption that the reading of 198mg of blood alcohol by the chemist is correct. In reply, DW3 said

That concentration of alcohol in the blood is enough to make a hardened drinker unfit to be in charge of a vehicle. He would however be unlikely to be drunk and disorderly and I would have thought very unlikely to defecate and micturate in his trousers. ... Incontinence happens in people who are very ill from alcohol. They will soil their pants. In the case of hypoglycaemia where there is soiling of the pants this is so rare that I have never seen a patient with whom I have been dealing who soiled their pants in this way. But I do not normally see patients who are having fits from hypoglycaemia. Convulsions are the commonest cause of previously healthy adults soiling their pants.

E

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DW3 was referred to pp 492–493 of exh D97 which sets out the characteristic features of hypoglycaemia. They are as follows:

- (1) The behaviour, if offensive, will be out of character with the standards of conduct of the past.
- (2) The abnormal behaviour will tend to appear suddenly or explosively.
- (3) It is likely to be unexpected and inappropriate to the circumstances and where the behaviour is anti-social or criminal there will be no evidence that it is planned or pre-meditated.
- (4) What is done will be senseless or at least poorly motivated. Confused or irrational thinking may be discernible.
- (5) No convincing attempt will be made to conceal anti-social or criminal actions.
- (6) The period of abnormal behaviour will be relatively brief, being measured in minutes or at the most an hour or so but certainly not in days or weeks.

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- A (7) Finally, there will be a period of amnesia surrounding the abnormal behaviour. It is possible for 'islands' of partial memory to be present during such a period of amnesia but these will be vague memories of important events.

In commenting on exh D97, DW3 said:

- B What is stated at p 492–493 remains true. I am confirming this insofar as hypoglycaemia is concerned. I do not want to talk about the other illnesses that have been mentioned.

- C He said that he does not believe that hypoglycaemia occurs in well-defined stages in terms of its effect on the brain. He added that a hypoglycaemic situation is normally at a reading of 3.5ml and that he would not classify a figure of 3.9 millimoles as diagnosing hypoglycaemia. He was then asked whether DW2's evidence is significant when he said that at a reading of 3.9 millimoles, the accused had certain symptoms and recovered dramatically after administration of glucose. In answer to that, DW3 said:

- D It is a significant finding because glucose is the specific treatment for neuroglycopenia which I distinguish from hypoglycaemia. The recovery after taking glucose means his brain was short of glucose. That would invite investigation as to why he was suffering from neuroglycopenia.

- E On being asked whether the accused was feigning amnesia, DW3 said:

- F I have no reason to believe that amnesia was feigned but I am open to persuasion. There is every reason to believe that the amnesia is true. I was particularly impressed by the account given by the accused of his first meeting with his parents when, if what he said is believed, it came as a genuine and real surprise to him. The surprise is that he had committed the offence. In my experience with hypoglycaemia patients they find it very hard to believe when told of the events that had occurred while they were suffering from neuroglycopenia. When confronted with what they consider to be overwhelming evidence they accept what is said to have happened.

- G DW3 was then asked whether, on the assumption that the bizarre incident on that day was brought about by hypoglycaemia/neuroglycopenia, the accused could have got into that situation without being aware that he was going through certain impediments progressively. In reply, he said:

- H Yes. This is exactly what happens in the condition that I call sub-acute neuroglycopenia which is now better known to experienced diabetic specialists as hypoglycaemia unawareness. It was accepted by the Court of Appeal in Britain in a case where I gave evidence as a true entity and that the warning symptoms that alert a diabetic or other patient to the impending brain mal-function is absent. Sub-acute neuroglycopenia is a mal-function of the brain that comes on gradually as hypoglycaemia develops. It is a syndrome distinct from acute neuroglycopenia with which every medical practitioner is familiar. In acute neuroglycopenia the patient experiences the symptoms of anxiety, trembling, rapid heartbeat, sweating and many other distressing symptoms. They are premonitory evidence of impending more serious brain mal-function due to increasingly severe hypoglycaemia.
- I

In his evidence on preserving the integrity of blood samples, he said that: **A**

... it must be preserved after collection unless the analysis is done immediately. There are various substances that can be used to preserve the blood. Normally for glucose we use sodium fluoride. It is also common to use potassium oxalate to prevent clotting. If the blood is to be analysed 48 hours later preservative must be added. If there is no preservative added the result is meaningless. The deterioration starts from the time the sample is collected. Keeping the sample in the refrigerator without preservatives slows down the process of degradation but does not prevent it. The result would be meaningless. **B**

In his cross-examination, DW3 was referred to the symptoms of intoxication when the blood alcohol level is 180mg to 300mg. When questioned on the symptoms at that stage, he said: **C**

The table is just a rough guide. If the alcohol level is 180–300 (DPP reads from table) all of those things can happen or none. At this stage of alcohol, the person is conscious. The effect of alcohol varies from individual to individual. A person who is intoxicated may become aggressive and commit murder. I have come across hypoglycaemia patients committing murder. It is fewer compared to murders committed by people when they are drunk. **D**

DW3 was then referred to para 67 of exh D84 where he had calculated the blood alcohol level in the accused at 8pm at about 40–80mg. He was then asked what the level of alcohol in the accused would be based on the level of alcohol level of 198mg in him at 1.30am. In reply, he said: **E**

I do not know. There is a guide to calculate it. But it is a bad guide. It could be the same, it could be higher or it could be lower. In this case drinks were available to the accused till 7.30pm. Alcohol goes from the stomach into the small intestine and then into the blood and unless we know for certain that all of the alcohol has been absorbed, we cannot be certain whether his alcohol level is going up or going down or staying the same. **F**

It was then suggested to him that his conclusion at para 67 could be wrong. He replied:

I accept it. Para 67 is based on an assumption. The blood alcohol of 198 milligram contradicts this assumption. I have never said that the accused's alcohol level at 8pm was 40–80mg. The assumption of 40–80mg is wrong. It is misleading. **G**

He said that a person heavily intoxicated will have a blackout. With such a blackout there can be amnesia. Having said that he has not practised as an alcohol clinical specialist, he added: **H**

I am familiar with alcohol symptoms only under experimental conditions. I have never given anybody enough alcohol for him to have a blackout. I am not going to give an opinion on the role of alcohol in this case except in relation to possible hypoglycaemia. In this case I was presented with a question about hypoglycaemia. I did not feel it was possible to discuss hypoglycaemia without reference to alcohol in which I have done substantial research. **I**

He said that it would be impossible for him to discuss hypoglycaemia without taking alcohol into account although he is not an expert on the

A clinical manifestation of alcohol. He was then cross-examined on hypoglycaemia caused by insulinoma. He was then told that in this case there is no evidence to show that the accused had undergone a stomach operation nor did he have any tumour in the pancreas. He was then asked:

B Without any investigation as to the cause of the hypoglycaemia can you safely say that the cause is from insulinoma?

He replied:

Insulinoma remains one of the possible explanations by Dr Ridzwan. He did not say any cause. I cannot say that the accused has no tumour in the pancreas.

C He was then asked by me:

Assuming there is no tumour?

He replied:

D Then there may be other causes of hypoglycaemia. In para 98, I am referring to insulinoma. Insulinoma is the commonest cause of hypoglycaemia due to an abnormality of insulin secretion. There can be abnormality of insulin secretion without a tumour in the pancreas. But it is less common than insulinoma. Abnormality of insulin secretion can occur with abnormal B cells which are not present in the form of a tumour. This condition is often referred to as adult nesidioblastosis. In the present case, no investigation as would satisfy me were carried out on the accused to determine the nature and cause of his purported hypoglycaemia.

F DW3 was then referred to the provisional finding of low blood sugar of the accused by DW2 and to exh IDD83. The cross-examination runs as follows:

G Q: Can you by this provisional finding say that the accused is having hypoglycaemia?

A: I cannot say conclusively. But it is suggestive.

Q: Without more can you confirm that the accused has some sort of hypoglycaemia?

A: I cannot say anything beyond the fact that I am surprised that further investigations were not carried out to determine whether the accused has an illness capable of producing spontaneous hypoglycaemic attacks.

Court: Did you not consider it necessary to conduct some tests before preparing your report?

H A: I was told that the defence could not carry out investigations but the prosecution could. If I had carried out tests I would have been much happier.

I He was then asked whether in this case the accused had fallen into a coma state. He said:

The only time in which coma may have occurred which was reported in the accused, was in the golf club incident where he collapsed.

DW3 then said that in this case it is possible that the accused was experiencing reactive hypoglycaemia. This type of hypoglycaemia first manifests itself between two and three hours after commencing to eat. He was then asked the probable time the accused would have had the first symptom of hypoglycaemia, if in fact he was undergoing an episode. In reply, he said,

A

It would have started after 3.30pm. Probably at 4.30pm. It had to develop before 8pm.

B

When asked about the possible time the accused could have recovered without treatment DW3 said that it would be impossible to say without knowing the cause of the hypoglycaemia. He added that he has no evidence to say that the accused was ever comatose and added:

C

Nobody told me what his mental state was at that time.

When asked whether he had not read the notes of evidence, he said:

He was snoring.

D

When asked whether at the time of his arrest the accused could show signs of staggering, slurred speech, palpitation and aggression, DW3 said:

I do not think that this is due to hypoglycaemia at that time.

When asked whether he agrees it is in line with intoxication, DW3 said:

E

It is consistent with blood alcohol of 198. I think it is unlikely that he was completely sober.

He was then asked whether in a hypoglycaemic situation automatism is uncommon. In giving his answer in the affirmative, he said:

F

Most people who experience hypoglycaemia do not go into automatism. Some do. They are a minority. It is not confined to people with diabetes. Any patient who suffers from any cause of hypoglycaemia may go into a state of automatism. The ones most likely to get it are those who have not been through a period of acute neuroglycopenia which can happen.

G

He said that those who are in a state of hypoglycaemia automatism are also suffering from sub-acute hypoglycaemia. DW3 was then questioned on the incontinence of the accused in this way:

Q: In this case there is evidence to show that accused had no epilepsy or seizure or convulsion?

H

A: I have heard no evidence of anybody saying he had a convulsion.

Q: In that event is his incontinence consistent with intoxication?

A: It is consistent with gross intoxication.

Q: The fact that immediately prior to being brought to the police station the accused was apprehended and assaulted — did it contribute to the incontinence?

I

A: It could have been.

- A** DW3 was asked whether, in light of the fact that accused was not involved in any accident, the incident that happened was done with a conscious mind. He replied:

I do not know what went on in his mind.

- B** When suggested that the accused was not undergoing any hypoglycaemic episode at that time, DW3 said:

I have no glucose measurement. I cannot say he was not suffering from hypoglycaemia or he was suffering.

- C** In his re-examination, DW3 said that this case does not show a pattern of alcohol-induced hypoglycaemia as there must be fasting for 24 hours in such cases. He said that reactive hypoglycaemia is a possibility as it happens in response to eating. He also said that the symptoms experienced by the accused in the hospital may be consistent with intoxication though the same presentation could be consistent with hypoglycaemia. He was then asked whether in the cases where he has appeared as an expert he was involved in the clinical examination of the accused. He replied:

- D** No, that is the role of professional experts. These are people who examine and look after patients. Persons like me merely comment on the data available and help to advise the court. I have never been rejected in the UK Courts.

- E** When explaining his answer in cross-examination that amnesia can be consistent with intoxication, DW3 said:

The manifestation of alcohol intoxication is not my field of expertise. When I said that amnesia can be consistent with intoxication that was not my field of expertise.

In concluding his re-examination, he said:

- F** With whatever materials provided to me I cannot conclusively say that accused did not suffer from hypoglycaemia in relation to his visits to doctors earlier. They were suggestive of hypoglycaemia. The evidence is not inconsistent with the suggestion that accused was suffering from hypoglycaemia. The accused had amnesia. That is because the accused told me. I would have thought that 1 in three patients presented with the case like that of accused would turn out to be hypoglycaemia.
- G**

- H** DW7 qualified from the National University of Ireland in medicine with a MBBCh degree in 1986. She then entered a training programme in internal medicine in the University of Cork Hospital. During this period she had six months working experience in endocrinology. Having obtained her Membership of the Royal College of Physicians, she started to specialize in the area of endocrinology. She did a one year research fellowship at the Mayo Clinic, Rochester. She has worked in the endocrinology department of the Universiti Kebangsaan Malaysia and is currently attached to the Lam Wah Yee Hospital as an endocrinologist.

- I** She had read the evidence given by the witnesses from a document similar to exh D92. She gave a fairly good account of the evidence in the case. She was aware of the medical condition of the accused prior to the incident. As she said:

From my readings of testimonies given to me, I know of four episodes between 1991 and 1995. Out of these four, the accused went to Pantai on three occasions and one episode happened in a golf course in Genting.

A

Having given further details of the episodes, she added:

Dr Ridzwan noted Mr Lee's blood sugar was less than 4. He administered glucose to Mr Lee and Dr Ridzwan noted Mr Lee's symptoms were relieved by the administration of glucose. The blood reading was less than four. But that is not absolutely important to determine the existence of hypoglycaemia. The important thing is the presence of the symptoms at the time that the blood glucose was 3.9 and the relief after taking glucose.

B

In testifying on the symptoms of hypoglycaemia, she said that they are very variable and they are not exclusive to hypoglycaemia. She said that hypoglycaemia is rare and it is known that the episodes can be episodic. Thus, the symptoms of hypoglycaemia can reappear after several years. She then said:

C

On the assumption of the accused having had hypoglycaemia on 22 August 2000 and the underlying cause is not discovered, there is a high probability of recurrence in the future.

D

When asked to explain this statement, she said:

What I meant by that is it could happen. He could have a hypoglycaemia attack in the future. When I used the term 'high probability', it was a mistake. It could happen or it may not happen. It can even be more serious if and when it recurs.

E

She said that it is not necessary for a person to go through different stages of hypoglycaemia as it is not a production line. A person can go into an aggressive presentation straightaway. This is because people with hypoglycaemia can immediately go into the neuroglycopenic stage without any warning that they are going into that stage. She said that even a person with normal medical results may have hypoglycaemia. In support of this she referred to a patient whom she had examined. As she said:

F

There was a patient in our hospital in 1999. In June 1999, she had palpitations and chest discomfort. No blood sugar was done. In late June she felt better but felt giddiness. Her blood sugar was 3.1. In October 2000, she felt palpitation. Her blood sugar was 3.6. She was given gastric medication. In March 2001, she presented with chest pain. Her blood was normal at 4.1. In late March, she attended for a follow-up. Her blood sugar was 4.7 which is normal. The doctor felt that in view of her symptoms and the low blood sugar earlier referred her to me to outrule any endocrine abnormality. At that time I took her history. I found her to be normal. So I decided to proceed with the prolonged fast test. In this test, a patient is fasting and we try to induce hypoglycaemia in the hospital. We also see if by inducing hypoglycaemia the patient will develop the symptoms. This test is designed for people who are suspected to have a disease of hypoglycaemia. The test is carried out by up to 72 hours of fasting and if there are no symptoms or the blood sugar does not go abnormally low of less than three then it is unlikely that that person is suffering from hypoglycaemia. When the lady underwent this test, she became hypoglycaemic. Her blood sugar fell to 1.9. At that time she was awake but less responsive to my questions. She looked as if she was day dreaming. She

G

H

I

- A** did not experience palpitations etc that she had experienced before. We operated on her and found to have insulinoma. That is one of the many causes of hypoglycaemia.

- B** She said that idiopathic hypoglycaemia, that is to say, people who are hypoglycaemic but the cause is not known despite investigation, accounts for 10% of hypoglycaemia patients. In the other 90%, the cause can be ascertained. With regard to amnesia in hypoglycaemia cases, she said:

Amnesia can happen in a case of hypoglycaemia. I do not think it can be said to be common. But it can happen. It has been known to happen. In a study of 60 patients with insulinoma 53% suffered amnesia of the episodes.

- C** In commenting on the relationship between hypoglycaemic unawareness and amnesia, she said:

I cannot give you figures but it is possible for a person with hypoglycaemia to be carrying out an act or action and be not aware of it at the time he was behaving that way. This is known in the world of medicine. Amnesia does not necessarily have to be its aftermath because amnesia means you do not remember. It does not mean that you were unaware of what you were doing at that time. Amnesia can be an aftermath of hypoglycaemia unawareness.

D

She was then asked this question:

- E** Looking at the behaviour accused exhibited during the episode bearing in mind that there was propensity of the accused getting low blood sugar, would you say that his conduct on that day was consistent with the conduct of a hypoglycaemia patient?

She answered it this way:

No, it is not typical but it is possible.

- F** She was then asked whether the conduct of the accused is consistent with intoxication or hypoglycaemia. In reply, she said:

- G** In answering that question I consider that the only laboratory test done showed he had a blood alcohol level of 198 at 1.15am and using what I term common sense his blood alcohol level would be higher at 8pm. If his blood alcohol was at a level that causes urination and defecation he would be stuporous. He would not be able to walk normally, up and down the road, his speech was noted to be normal, he did not have any shaking in his hand, but the taxi driver said he appeared strange. Those features suggest that he may be hypoglycaemic at 8pm. Between the two hypoglycaemia is more likely because the only way to confirm with 100% accuracy that it is hypoglycaemia is to have a measurement of the blood sugar at the time of the incident. It is difficult to get such measurements.

H

When asked whether there is evidence in this case with regard to the seven characteristics listed in exh D97, she said:

- I** My reading of the notes of this case shows that Mr Lee was observed to behave normally at the restaurant and getting into his car. The abnormal behaviour was noted at 7.55pm when he stopped his car and attempted to confront a motorcyclist who sped away. He has amnesia from sometime at 4.30pm. But the director noted no abnormal behaviour. My understanding is that the

abnormal behaviour started at the roadside and lasted till he was arrested. Amnesia is different. When he was taken to the hospital he had partial recollection until 5pm because that was when he realized he was arrested. The seven criteria have been more or less met in this case. ... I do accept the definition of the existence of automatism as laid down by Glasgow and that the events of 22 August 2000 fulfill these criteria.

A

She concluded her examination-in-chief by saying that she found the classification of hypoglycaemia into acute and sub-acute neuroglycopenia by DW3 as being difficult and confusing. She is familiar with the symptoms of hypoglycaemia as being neurogenic and neuroglycopenic. In further elaboration, she said:

B

The neuroglycopenia symptoms are due to the discharge of the autonomic nervous system and in this article the symptoms assigned are warmth, anxiety and nausea. The neuroglycopenia symptoms are dizziness, confusion, tiredness, difficulty in speaking, headache, inability to concentrate. With regard to neurogenic symptoms they may also be shaking or tremoriness, pounding of the heart, nervousness or anxiety, sweating, hunger of tingling to the autonomic group. The neuroglycopenia group has feeling of warmth, weakness, confusion, difficulty in thinking and fatigue or drowsiness.

C**D**

She said that it is the neuroglycopenia symptoms that produce unawareness and automatism. In her opinion the symptoms experienced by the accused in 1991 are suggestive of symptoms related to the autonomic nervous system or the neurogenic symptom. As she said:

E

I feel that the accused in the early 1990s had persistent complaints of chest pains, heart palpitations, numbness of the hands, sweating excessively. He attended the Pantai Medical Centre. His blood sugar was low. His symptoms were relieved by taking glucose. The next morning he attended Dr Ridzwan's clinic complaining of chest pains, heart palpitation, numbness of the hands and sweating. His wife testified that he had breakfast at 7am. To me these symptoms in 1991 are suggestive of symptoms related to the autonomic nervous system or the neurogenic symptom. Prof Marks is an eminent researcher in hypoglycaemia. I am trying to simplify the classification of hypoglycaemia.

F

In her cross-examination, DW7 said that having read the testimonies of the witnesses in this case she believed that the accused could possibly have been suffering from a hypoglycaemic attack on 22 August 2000. In explaining that the symptoms of hypoglycaemia are not exclusive, she said:

G

Some of the symptoms are not exclusive to hypoglycaemia. It can also appear in the case of a person who is intoxicated. Aggressive behaviour is common to hypoglycaemia and intoxication. Also argumentativeness, uncooperativeness, tremor, staggering gait, slurring speech. Hunger is suggestive of hypoglycaemia. Paleness, sweatiness, nausea can happen in both. Hunger is also suggestive of intoxication. Drowsiness and incoherence can be seen in both. Difficulty in writing is also common. Difficulty in focussing is also common to both. Visual disturbance can occur in both. Tiredness and clumsiness can occur in both. There is no specific symptom that is unique to hypoglycaemia or to intoxication. Depression is more present with intoxication.

H**I**

A She said that as the symptoms of alcohol intoxication and hypoglycaemia are similar she would not be able to say from a physical examination alone that the accused's behaviour was due to one or other of the two conditions. She added:

B I mean that if Mr Lee was presented to me and I did not have the facilities to measure his blood sugar it would be very difficult to differentiate between hypoglycaemia and alcohol intoxication.

Her further cross-examination on the possibility of the accused having been intoxicated on that day runs as follows:

Q: On that day accused was suffering from alcohol intoxication?

C **A:** Accused had alcohol but alcohol intoxication cannot explain some of the evidence given by witnesses at the scene. If he was intoxicated he would not be able to drive a vehicle without incident on a strange route. This was not his normal route. And through two toll bridges. A person intoxicated is unlikely to have done that because his blood alcohol level would be in the region of 300 mg at about 7pm. This is my assumption.

D **Q:** Are you aware that during the drinking session the accused was a little bit talkative. Wasn't he affected by the drinks?

A: I was not aware of that.

Q: That was the evidence given. Is it not indication of alcohol influence?

A: Yes, it can be.

E **Q:** At about 8pm, do you agree that accused can also be said to be under alcohol intoxication?

A: Yes.

She said that in the case of neuroglycopenia symptoms the supply of sugar to the brain is so insufficient that it cannot sustain or fuel the brain cells. At that stage, the person will be in a coma. A blood test would show whether such a person was undergoing a hypoglycaemia attack. She added:

F

Without such a test on clinical grounds alone to differentiate with a 100% certainty between an alcohol induced and a hypoglycaemia induced coma would be difficult.

G She added that quite often when a person has hypoglycaemia symptoms it would be found to be not so when he gets up. A person in a severe neuroglycopenia stage and is not treated can wake up by himself without treatment as a result of the counter regulatory mechanism in the body to correct hypoglycaemia. As she said:

H In the case of a healthy person, the counter regulatory mechanism starts immediately. For a person to go into coma, it means that the consumption of alcohol is greater than the production of glucose in the body by the counter regulatory mechanism. I disagree that a person in a coma stage cannot recover spontaneously unless treated.

I She said that generally it takes about three to four hours for food to be turned into glucose. After that period, the liver maintains the blood sugar level by the process of glycogenolysis. This will go on for three to eight hours. In her cross-examination on the health of the accused, she said:

- Q: In the case of normal healthy person who had breakfast at 10am and a meal at 2.30 to 3pm and 3pm to 7pm, can he get hypoglycaemia? **A**
- A: In a normal healthy person, I would say no.
- Q: I believe you have read the notes of evidence. The accused can be considered as healthy?
- A: It is my opinion, the accused is not a normal healthy person because I do not consider his frequent attendance at Pantai Medical Centre in 1991 and his subsequent visits to Dr Ridzwan 1995–96 as that of a normal healthy person. He also had gastritis. **B**
- Q: Do you agree that a person with these kinds of problems will have symptoms of heart burns?
- A: It dramatises what they are experiencing. **C**
- Q: Dr Ridzwan's finding was provisional. Can these one or two occasions provisionally diagnosed be conclusively said that the accused is not a healthy person?
- A: The accused is not a healthy person. His history shows two episodes. The first was on 23 October 1991. I think the provisional findings are good grounds for me to say that accused is not a healthy person. **D**
- Q: When you say, good grounds, do you mean suggestive of hypoglycaemia?
- A: Yes.
- Q: When something is suggestive, does it not have to be proved?
- A: Yes. **E**
- Q: Can you conclusively say that by these two provisional findings that accused had reactive hypoglycaemia on that day?
- A: I cannot say conclusively as that would require a blood test at that time.

Her further cross-examination on the point of time the accused's hypoglycaemia would have started is as follows: **F**

- Q: If the accused's cause of hypoglycaemia is idiopathic and has lunch at 2.30pm, when do you think his reactive hypoglycaemia would have started?
- A: His lunch finished at 3pm. He continued to drink beer until 7pm. Hypoglycaemia may have occurred between 7pm and 7.30pm. This is because the food consumption finished at 3pm. **G**
- Court: What about the production of glucose by the liver?
- A: The food consumption finished at 3pm. By 7pm, the liver should begin to produce the glucose to maintain the blood sugar. If a person has hyperinsulinaemic then the liver cannot produce glucose because one of the actions of insulin in its function of reducing blood sugar is to inhibit glycogenolysis and also the process of gluconeogenesis. If a person does not have this problem and the liver is normal the liver will produce enough glucose. In saying that the accused may have had hypoglycaemia, I am relying on the symptoms and signs observed by the witnesses. I am not disregarding the glucose produced by the liver. This is because of other hormones involved. The liver does not act in isolation. It is influenced by the hormones that circulate in the body. That might slow down the production of glucose in the liver. **H**
- I**

A On this issue the further cross-examination is as follows:

DPP: When you said that the accused had hypoglycaemia at 7pm did you take into consideration the part played by the hormones?

A: Yes.

B DPP: If that be so, do you agree that in that event the episodes would have been at 9 or 10 o'clock?

A: I disagree.

DPP: I further say that the accused did not suffer the hypoglycaemia episode at about 7pm?

C A: I disagree because my interpretation of the events is that the accused suffered a hypoglycaemia episode that began at 6.30 to 7pm with the blood sugar reaching its lowest point at about 8pm and the gradual recovery occurring between 10 and 11 o'clock.

DPP: You are making this finding without any clinical finding?

A: Yes. I was just asked to read the evidence and give my opinion.

D DPP: You are still sure despite that?

A: That is my opinion. I disagree that accused was under the influence of alcohol.

She said that she would classify the case of the accused as one of sub-acute hypoglycaemia. In such cases, it is possible for a patient to be unaware that he is having hypoglycaemia. Her further cross-examination of the physical condition of the accused on that day runs as follows:

E Q: Can a person progress to a fugue or acute or chronic neuroglycopenia?

A: The chronic neuroglycopenia that Prof Marks speaks about is a new disorder to me. He talks about a patient becoming demented. I do not know about that. A person who has sub-acute neuroglycopenia can progress into acute neuroglycopenia or coma.

F In this case I did not read of any evidence of a coma. When he describes his memory of his visit to the hospital that is suggestive of acute neuroglycopenia. At 8pm, he was in sub-acute stage and at 1.30am, he may have been in acute hypoglycaemia stage. I think at 1.30am, the symptoms of alcohol predominated.

G Q: Can it be said that at the police station the accused was recovering?

A: He may have been recovering.

Q: Can a person who has gone through sub-acute hypoglycaemia progress into acute hypoglycaemia immediately upon recovery?

H A: A person who experienced sub-acute neuroglycopenia during recovery may experience some of the symptoms of acute neuroglycopenia. It is unlikely for a person who is recovering from sub-acute neuroglycopenia to progress into acute neuroglycopenia.

Q: Have you ever experienced the reverse situation of a person claiming to be hypoglycaemia to be alcohol intoxicated?

A: I have never had such a case.

I Q: That is what that happened in this case?

A: I agree that at 1.20am the accused was acting as a person who was intoxicated.

Q: Do you agree that the blood reading of accused at 1.20am corroborates the evidence of Dr Bhawani?

A

A: I agree.

She agreed that the writer of exh D97 in saying that the seven characteristics apply in cases where the causes are known. She said that in order to be affirmative of a hypoglycaemia condition a 72-hour prolonged fasting test must be done. In this case, the accused was not referred to her. In her re-examination, DW7 was asked to explain her conclusion that the accused would have had his hypoglycaemic attack between 6.30pm to 7pm. The examination runs as follows:

B

In this case the hypoglycaemia would have come about 6.30pm to 7pm because the consumption of food was between 1pm to 3pm and from there on there was no consumption of food. Beer has little calorific value. The glucose from the food would have kept the accused going till about 6pm to 7pm. Thereafter, glucagon and the other counter regulatory hormones should have begun to act on the liver to stimulate the liver to produce glucose as well as the counter regulatory hormones.

C

Q: If what you have just described took place could you then explain your opinion about hypoglycaemia attack taking place in the accused from 6.30pm to 7pm?

D

A: This is because Mr Lee's behaviour between 7 and 8 o'clock suggest that the counter-regulatory mechanism failed to maintain his blood sugar level to enable his brain function to work. The symptoms and signs he displayed are in keeping with neuroglycopenia.

E

Q: Going by the symptoms he displayed at about 8pm are you saying that by using those symptoms you are expressing an opinion that the system that ought to work in fact failed?

A: Yes.

Q: Is that your opinion?

F

A: Yes.

Q: For clarity, could you tell us what is the system that failed on that day?

A: The cause of his hypoglycaemia has not yet been identified. So I cannot say that this failure was due to too much insulin or due to failure to produce glucagon or due to a failure to produce growth hormone plus glucagon or due to a failure of the autonomic nervous system. I have to say on the testimony that I have read that it is idiopathic.

G

Q: Isn't idiopathic after tests have been conducted and no cause has been identified?

A: Yes.

Q: Since you know that tests have not been done in this case why do you classify it as idiopathic?

H

A: Because if the tests were done to investigate the cause of the accused's hypoglycaemia he may become hypoglycaemic during the investigation and still have normal insulin glucagon, adrenaline, cortisol and growth hormone levels despite becoming hypoglycaemic.

I

Q: That is after a test?

A: Yes.

- A** Q: Do you still want to classify it as idiopathic?
- A: I do not know what the cause is. The cause is unknown rather than idiopathic.
- There must have been a failure of the liver system to correct the falling blood sugar because he demonstrated signs of neuroglycopenia. The liver is the predominant organ. The kidney produces only 15% of the requirement. That may help in preventing permanent neurological damage or death in hypoglycaemia.
- B**
- A liver may be structurally normal but it must also function normally. A liver will only function normally under the influence of the hormones that are produced in the pancreas, the adrenal glands and pituitary glands. The liver is influenced by the neurotransmitters from the autonomic nervous system. The liver can function properly only if the hormonal system and the nervous system are in tandem. In most hypoglycaemia cases the failure is usually in one of the symptoms I have described.
- C**

Her final words on the medical condition of the accused are as follows:

- D** Using the normal physiological response the unawareness of the accused may have begun from 7 o'clock until sometime between 10 and 11 o'clock. Based on the evidence he came out of it completely the next morning. Presumably because he was given some breakfast. In the state of unawareness he would not know what he was doing.
- E** Going by the medical reports on accused there is strong suggestion of hypoglycaemia. This is considering the medical reports and testimony of witnesses of what they saw. When I said that it is possible that accused had hypoglycaemia I mean that from the evidence presented to me (the witness at the scene, the witness at the lunch, the evidence of the arresting officer) I believe that Mr Lee's behaviour at 8pm is more suggestive of hypoglycaemia than alcohol intoxication.
- F** I maintain that the seven criteria in D97 have been met in this case.
- Dr M Mahadevan ('DW9'), a psychiatrist has very impressive qualifications as listed in exh D102A. His report on the accused is exh D102B and he gave oral evidence clarifying certain features of the report. The material parts of exh D102B read as follows:
- G** 1 Bearing in mind that the issues and references raised herein relate to the charge of murder brought against the accused I have been asked specifically to consider the following:
- (a) was the accused labouring under alcohol intoxication as the cause of his conduct at the time of the incident;
 - (b) was the accused suffering from the effects of hypoglycaemia/neuroglycopenia as the cause of his conduct at the time of the incident;
 - (c) any other discernible cause/s of the conduct of the accused at the time of the incident.
- H**

- I** In considering the above I have on my own accord also considered whether any of these factors could have been working together rather than independently. In any case my main psychiatric investigation was towards determining the cause/s of the rather bizarre behaviour of the accused at the incident that led to the unfortunate death of the victim.

(d) in relation to both alcoholic intoxication and/or hypoglycaemia (or any other causes) I have been asked to investigate and express my opinion on whether:

A

(i) the accused was aware or conscious of his acts or conduct at the time when the questionable acts or conduct were being performed by the accused at the incident;

(ii) the accused suffered amnesia following the incident and in this regard to consider if the amnesia was genuine or feigned;

B

For the purposes of my opinion in this case, I have relied exclusively on the following:

(a) my oral attendance in court from commencement of the trial until now. I have made observations and notes of the subject matter of the evidence adduced by each one of the witnesses for both Prosecution and Defence and the exhibits they have produced. I have taken special note of the accused's testimony in court, the demeanour he had exhibited while giving evidence in court and generally when he was sitting in the accused's dock observing the proceedings. I was able to make special observation of the accused (in both isolated and comparative terms) in court as I had observed him on many occasions before when I interviewed him in prison, or in court, prior to the trial;

C

(b) whenever I needed to double-check the facts of the case as unfolded in the court I have often sought assistance from the notes of proceedings provided to me by the defence lawyers. I believe a copy of these notes are already placed as an exhibit in court;

D

(c) my interviews of the accused, more than 26 times in the remand prison in Sungei Buloh. These sessions lasted between 1½ to 2½ hours per session. I have also conducted interviews of the accused whenever there was opportunity on occasions when the accused was produced in court. My earliest contact of the accused was on 30 August 2000 when he was produced in court to be charged. After he was charged, I spent a total of three hours with the accused. I made vital preliminary observations and investigations of the accused.

E**F**

For purposes of conducting my interviews with the accused prior to the start of the trial I had to resort to reading or being briefed of the various statements ('initial materials') of the witnesses such as that of the accused, his wife Noor Salasawati, his parents YB Dato' Douglas K K Lee, YB Datuk Ruby Lee, his sister Janet Lee, the physicians YB Datuk (Dr) Ridzwan Bakar, Dr Thiagi Selvanayagam. But these statements were merely used as background materials for me to investigate and conduct my interviews of the accused. I have personally interviewed the above personalities on several occasions. In this opinion, however, I have been strictly reminded that I should only consider the evidence as unfolded in court and as such I have attempted in this opinion to provide answers to the issues raised by making reference only to what transpired at the trial. In this case we are fortunate because what has been produced at the trial is far more complete than the initial materials I was provided with and as such my reliance of the materials uncovered at the trial itself provided very satisfactory basis for my opinion.

G**H**

In the course of my interview, I have managed to extract various information and facts from the accused which I will endeavour to explain to the court in coming to certain conclusions about the accused's personality, and on the

I

- A** issues of the unawareness of the events, and the amnesia suffered by the accused after the incident. The question of whether the amnesia is feigned or genuine is also determined by certain tests and factual extraction from the accused during the interviews as well as from the evidence revealed in court. These will perhaps be the only areas where extrinsic facts (as opposed to those discovered in the court) are being used by me in my analysis and opinion.
- B**

With regard to the medical history of the accused exh D102B reads as follows:

I have discussed with his general practitioner Dr Thiagi and his personal consultant physician Yang Berbahagia Datuk Dr Ridzwan Bakar.

- C** He has been diagnosed as likely to be suffering from episodes of hypoglycaemia. However, the underlying cause is yet to be ascertained. He has been advised to take glucose drinks regularly and whenever he is aware of impending hypoglycaemia (if that is possible). He always associates hypoglycaemic attacks with 'heart attack' as in both cases, the signs, symptoms manifested are almost identical and simulate each other. Despite assurances, he has this mortal fear of having an impending heart attack. Consequently, he fears he may precipitate a heart attack in prison if he misses his glucose drink, where timely crisis intervention is not feasible or instantly available. 'Cardiac Neurosis — De Costa's Syndrome'. This is further strengthened by the fact that despite being told way back in 1991 that the symptoms he suffered although seemed like a heart attack was in reality a hypoglycaemic attack. His subsequent hypoglycaemic attacks were always considered as 'Imminent Heart Attacks'.
- D**
- E** He is also on antacids (Novalucid) for gastritis and dyspepsia.

In dealing with the terms of reference, DW9 goes on to state in exh D102B as follows:

- F** Behavioral Chart for Drunkenness

- 1 Dry and Decent
- 2 Delighted and Devilish
- 3 Delinquent and Disgusting
- 4 Dead Drunk

- G** This behavioural chart is taken from a known book 'Guide to Psychiatry' — by Myre Simms — accepted in the Esther Chan murder trial before Justice Syed Othman on September 1970 in Johore Baru High Court. From Miss Lim (PW3), the taxi driver (PW4) and the star photographer (PW6) we can only go by behaviour as we do not have the blood alcohol level taken at the point of the incident.
- H**

- I** When a person drinks, the 1st stage is being dry and decent. This means he is sociable, pleasant, amiable and affable. If he takes more drinks he then goes on to the second stage of being delighted and devilish. This is where he becomes euphoric, cheerful and gregarious. If more drinks are consumed, he then becomes intoxicated or inebriate. He is going into the third stage, where he is delinquent and disgusting; this is the stage when he is drunk. He would be anti social or delinquent or belligerent. If he takes more, he goes into the fourth stage of being dead drunk. In this stage he would fall asleep, go into a coma and eventually ensue in death.

Looking at the case at hand, from the testimony, which has been adduced by Dr Bhawani, the accused sat in front of her, and was responsive to her questions. He was heard to be verbally abusive but not to her. In any case it was to find out why he was arrested. He was able to write a note P17 with certain clarity. His handwriting was steady, bold and discernible although in handcuffs. A person under the influence of alcohol would have writing close to scribbling or a scrawl. His behaviour at this time would if anything falls under stage one of Dry and Decent or at most stage two of being Delighted and Devilish and accordingly not drunk.

A

B

According to the testimony of Inspector T Sivakumar and the Investigation Officer, ASP Wan Kok Chai the accused had defecated and micturated after his arrest but the exact time has not been established. If the defecation and micturition were caused by alcohol, this would be in line with the 4th stage of the behaviour chart ie Dead Drunk. Defecation and urination if caused by alcohol, would indicate that the accused was already in 'dead drunk' category, which could proceed to a comatose stage or death.

C

At the police station, he had apparently snored and slept for 20–25 minutes and woke up.

If he was intoxicated to such an extent to have defecated, it would be highly unlikely, that he would be able to wake up especially within 20–25 minutes. He is also highly unlikely to have dramatically moved from 4th stage behaviour to first or second stage behaviour in a relatively short interval of time ie when he was before the Investigating Officer or the Doctor.

D

The testimony of Noor Salasawati indicated that the accused never snored. The 'snoring' sleep of the accused, in my opinion was in fact a quiet convulsion (an epileptiform-equivalent attack — but not actually epileptic).

E

The evidence adduced so far states that the accused drove a length of 12km from the restaurant to the place of incident. He was able to manoeuvre through the narrow lanes leading out of the restaurant going through a labyrinthine maze of roads. The accused drove with no reports of accidents, mishaps or misbehaviour all through the 12km. The accused was able to pass through two toll plazas, which required payments at each toll gate. The accused was said to have operated his hand phone to call his wife whilst driving without any mishaps at about 7pm or so.

F

The activities mentioned above are complex activities by any measure, which required coordination and reflexes to operate dexterously. If the accused was so intoxicated, he would not have been able to conduct these activities without any mishaps. Medical science now know that a person undergoing hypoglycaemia can carry out complex activities without being detected by a third person as unusual.

G

In further explaining that a person under the influence of alcohol will not be able to carry out complex activities, DW9 said:

H

When I used the words 'under the influence of alcohol' it means that he is intoxicated to the degree that he cannot do these complex activities. In terms of my behaviour chart, it refers to a person in the third stage and beyond.

DW9's report then continues as follows:

I

It is also a known fact that an intoxicated person will always find his way home, despite it being a hazy memory (if at all) as to how he got back home.

- A** In this situation the accused on his way home, was lost and in fact landed up on a road, which was in a completely different direction.
- The accused's actions were most bizarre and completely out of character with the standards of conduct of his past at approximately 8pm on 22 August 2000.
- B** If the bizarre incident was due to alcohol intoxication, the blood alcohol of the accused must necessarily and correspondingly be at its height to explain this stage three of Behaviour chart. Assuming that the blood alcohol was at its highest at the time of incident, this flies in the face of testimonies of the eye-witnesses, where the accused apparently walked straight, did not have a staggering gait etc. He was not slurring. These are signs of an intoxicated person. The accused lacked these signs. Three eye-witnesses' testimony confirm his behaviour and gait. His manner, and behaviour at the time of the incident is not consistent with a person under the influence of alcohol. The answer to his behaviour at the time of the incident must be something else. This is virtually certain.
- C** According to the chemist's testimony the accused had gun powder residue on his 'left web'. This indicates that the accused probably would have used his left hand to shoot. The accused is a right-handed person. This was confirmed by testimonies from different witnesses. Unknown to the accused, I myself conducted a test by offering a pen to the accused. I purposely directed the pen towards his left hand. The accused despite being in handcuffs took the pen with his left hand and adjusted his hands in the confines of the handcuffs to move the pen from the left hand to the right hand to write. Such pattern of behaviour is never heard in alcohol cases. It does indicate that the Central Nervous System has been temporarily affected for the brain to have switched from the dominant left side to the right side of the brain. Such behaviour is not consistent with alcohol. On the other hand, such behaviour can happen in hypoglycaemia cases.
- D**
- E** The final reason as to why alcohol is not applicable in this case is that evidence has been adduced that the accused can consume Ω to 1 bottle of whisky on rare chosen occasions. He is a social drinker of moderate to high level. He has been drinking for 30 years. He is tolerant to hard liquor and beer. My experience tells me that he has built certain amount of tolerance for alcohol. The evidence adduced states that he had consumed approximately six beers that afternoon (a very modest amount to him). There is nothing to suggest that his tolerance level has gone down just before the incident. His
- F** medical examination suggests that all his vital organs were in perfect order. It is a known thing that tolerance to alcohol can gradually go down in an individual due to: (i) deterioration of certain organs; (ii) age. Neither applied to the accused. In any case, alcohol tolerance can only gradually go down — never suddenly!
- G**
- H** At the hospital at 1.15am on 23 August 2000, the accused's blood sample gives reading of 198mg/100 millimoles. This reading is not substantiated by clinical findings. In my experience the following tests should have been conducted. There are:
- (i) walking on a straight line test;
 - (ii) finger nose test;
 - (iii) intention — tremor test;
 - (iv) Nystagmus test, there was no record in her notes that she had conducted it although Dr Bhawani claimed she had done it;
- I**

- (v) stand on one leg at a time and to see if he can balance;
- (vi) memory test to check if the accused was oriented to time, place and person; and
- (vii) Babinsky — or Plantar Reflex.

A

He was apparently not orientated to place, because he was under the impression he was at University of Malaya's University Hospital (U.H.). He had thought that because Dr Bhawani knew of Prof Ernest Yeoh he must have necessarily be at U.H. The accused did not know that Prof Ernest Yeoh had been transferred from UH to Kuala Lumpur General Hospital.

B

He was furthermore not orientated to time, because he wrote in P17 that he had been arrested at 5pm when in reality he was arrested at about 8.20pm.

C

He was also not orientated to person as he thought that the Investigating Officer Wan Kok Chai was actually Khoo Ah Si, a former friend who was also previously in the police force, a long retired police officer.

The accused was genuinely disorientated to time, place and person.

Blood alcohol should and could not be interpreted except in the light of clinical findings. Thus, I am of the opinion that the incident that took place on the 22 August 2000 was not due to alcohol and is more consistent with hypoglycaemia.

D

Amnesia

The accused claims to have amnesia of the incident. His memory of the day of the incident lasts up to the time he was lunching with his friends at the restaurant. His next memory is waking up in the police station.

E

I am of the opinion that the accused's amnesia is not feigned/malingering.

I have had prolonged psychiatric examination with the accused over the span of this last one year.

The hours of interview run up to over 60 hours in 25 visits. In all my visits, the accused has always been consistent both in his memory of the facts surrounding the case and of the details. He has been brutally frank and honest. He has never evaded difficult and hard questions pertaining to the incident. He is possessed with remorse and chagrin. He is riddled with guilt. He is frustrated by his inability to remember the events that took place. This frustration has made him depressed and is now suffering from Post-Traumatic Stress Disorder (PTSD) 309.81 — DSM IV (Diagnostic Statistic Manual IV) of the American Psychiatric Association (APA).

F**G**

The accused has never shown any exaggerated behaviour or silly behaviour. I had brought to his attention to a strong local Chinese folklore belief that on the alleged day 22 August 2000, it was the day of the hungry ghost. The hungry ghost will wander around and it possesses vulnerable personality who comes under its influence. It will instigate them to do anti-social actions. I reminded him that where the incident took place, is near the biggest cemetery. So I asked him whether he would have been possessed by this spirit of the hungry ghost and in consequence he is not able to remember. He vehemently and frequently refuted my suggestions. He did not seek refuge under this umbrella. He could have conveniently claimed refuge that he was a victim of this superstition and consequently justify that he had no memory of the incident. He also told me that his wife is a devout Muslim and she too does not believe in this Chinese cultural belief.

H**I**

A From his demeanour, I could notice that he was genuinely puzzled and bewildered by the events that had taken place on the evening of the 22 August 2000.

I had conducted several tests on him to rule out Ganser's syndrome.

B Ganser's syndrome/Prison psychosis: The patient being under charges from which he would be exonerated were he irresponsible; begins, without being aware of the fact, to appear irresponsible. He appears stupid and unable to comprehend questions or instructions accurately. His replies are vaguely relevant to the query but absurd in content. He performs various uncomplicated familiar tasks in an absurd manner or gives approximate replies to simple questions.

C I conducted the first test on 11 January 2001. I asked him to do simple maths. I also asked how many legs does a dog or horse have. The answers he gave were always accurate and never approximate. I conducted a similar test on 29 October 2001, again he gave accurate answers. He was consistent and did not exhibit puerile conduct.

D Further in support of the fact that the amnesia was not feigned were the surrounding circumstances of the incident.

The evidence before the court has not revealed any motive whatsoever for the shooting. The shooting incident was not pre-meditated, or pre-planned. It was senseless and conducted in a non-structured, non-organized and non-directed manner.

E The incident took place in full view of the public and the law. There was no surreptitious behaviour or concealment by the accused at the time of the incident. It was done as to involve risk-taking conduct, jeopardizing his own life.

At the time of the incident, the accused was in the imminent danger of being shot or losing his own life. He had shot into the back of an empty army Land Rover. The army personnel if armed would have returned the shot. This behaviour of his was a suicidal-equivalent attack.

F The accused's manner after the shooting has to be noted. He did not escape or tried to run away. If he was aware of what he was doing he would have made some attempt to conceal the weapon or sought refuge or run away. His Volvo was still there and at the spot where it was 'parked'. Getting away was not difficult. It was apparent that he was at sea-confused-lost!

G There are certain other factors that led to the inevitable conclusion that the accused is not feigning amnesia. Looking at the evidence before us in court, from the time he was becoming aware, his responses and behaviour were consistent with a man who was confused and totally unaware of what was taking place viz his protests as to why he was in handcuffs, why he was arrested, abusive remarks, his need to seek his wife to inform her. His behaviour pattern was consistent all the time with an air of incredulity until he was told by his family of what transpired. He finally believed the accusations hurled at him and he broke down. This breaking down exhibited total disbelief of his situation. Till then he was in a state of bewildering puzzlement by his arrest.

H The accused enquired upon waking up in the cell, about his gun. This enquiry flies in the face of logic. It is an extremely audacious act of him to enquire about his gun if he was even remotely aware of the events the night before. His gun was used by him, which caused the victim's death. If he was aware, he would not have compromised himself in this culpable fashion.

I

Various acts of the accused, post the incident, were contemporaneous and showed not 'an afterthought'. For instance, he could have said to the doctor that he could not remember if he drank liquor or not. But he steadily admitted. He could not have known the significance of writing '5pm' as his arrest time on P17 when the evidence against was overwhelming. He could not remember even facts that were in his favour such as the calling of the Investigating Officer 'Khoo Ah Si'. He could not remember the public throwing stones at him or assaulting him. And very glaringly when the doctor asked him for his medical history, he did not even mention about his low blood sugar condition when this condition was already available to him as a possible defence even then. His amnesia is global especially nearer the incident itself. It is only at the tail end of the night of the 22nd and early 23rd that he began to have islands of memory.

I have had 41 years of clinical experience. Part of my training and experience is the detection of feigning, malinger or pretending. I have used my clinical acumen to assess the accused to the best of my ability. I have observed his demeanour in court and in remand prison and found that there is no reason to suspect amnesia was feigned.

Diagnosis

The accused suffered from hypoglycaemic cerebral insufficiency giving rise to metabolic encephalopathy. Metabolic encephalopathy is when the cerebral milieu is metabolically hostile to the normal function of the cerebral cortex as in hypoxia and hypoglycaemia.

I must and should stress that the hypoglycaemic cerebral insufficiency is not at all a disease of the mind but a temporary dysfunction of the mind directly related to glucose metabolism.

Hypoglycaemia

(a) Metabolism

Hypoglycaemia results from a disturbance in glucose metabolism leading to a depressed plasma glucose concentration. The characteristic clinical syndrome of hypoglycaemia includes alterations in mental function. All tissue requires constant supply of oxygen and of oxidizable substrate. Under normal conditions, brain metabolism is characterised by an exceedingly high consumption of oxygen and an almost exclusive dependence on glucose as a source of energy.

Glucose is the primary oxidizable substrate in the central nervous system. The central nervous system is dependent almost exclusively on glucose as a source of energy for metabolism. Rates of glucose metabolism are the highest in the cerebral hemisphere and parts of the cerebellum.

(b) Altered brain metabolism

A variety of changes, both inside and outside the central nervous system, result in secondary alterations of brain metabolism.

In hypoglycaemia, the brain does not store glucose, a carbohydrate that is the major source of energy in the body. Glucose is constantly used in the manufacture of compound that powers the nervous system.

In most people, the blood glucose level normally ranges between 80 and 100mg per 100 millimoles. A drop below 70mg per 100 millimoles often induces changes in sensation and behaviour, with some people experiencing alterations of mood and change in the level of consciousness at that concentration. Thought disorder, excitement and abnormal

A electroencephalographic activity may appear. Nearly 25% of the general population, however, routinely have blood glucose level below 50mg per 100 millimoles without concurrent symptoms of hypoglycaemia. Some people tolerate even lower transient levels without associated symptoms.

(c) Hypoglycaemia Unawareness

B Hypoglycaemia is a physiological condition related to the body. Hypoglycaemia unawareness is related to the central nervous system being defective or affected as a result of such condition. The sudden low level of glucose affects the central nervous system rendering the patient unaware of his onslaught of his hypoglycaemic attack.

The decrease or lack of glucose will normally evoke a characteristic hierarchy of responses to avoid development of hypoglycaemia.

C Initially at approximately 70mg/dl (4mM); there is secretion of counter regulatory hormones and a concomitant increase in the discharge of autonomic nervous system neurotransmitters. If these initial responses do not prevent a further decrease in the plasma glucose, further falling of plasma glucose level will bring out autonomic symptoms. The activation of the autonomic nervous system probably evolved primarily as an alarm. This means that the body will automatically provide more volumic blood to the essential organs of the body namely, the brain, heart and kidney. The body will drastically reduce its blood supply to the non-essential organs such as the limbs, gut and other big muscles.

D The much smaller volume of blood in the gut means that absorption of anything in the intestines is slowed down. This applies also to alcohol in the gut. This will possibly explain the reading of 198mg of blood alcohol at 1.15am on 23 August 2000 in spite of the fact that the last consumption of any beer would be before 7pm or so and only six glasses of beer were consumed in total.

E Failure to recognize the autonomic warning symptoms or failure for them to occur before the development of neuroglycopenia is what is commonly referred to as hypoglycaemia unawareness.

F (d) Cerebral Insufficiency

In the accused, the cause of his hypoglycaemia is at the moment not ascertained.

G The hypoglycaemia attack that occurred on the accused was so severe that it caused a severe 'cerebral insufficiency'. This means the lack of sugar/glucose caused the accused's cerebral milieu to be metabolically hostile to the normal function of cerebral cortex and thus he is said to have a metabolic encephalopathy.

(e) Metabolic Encephalopathy

H In the accused, the hypoglycaemic cerebral insufficiency gave rise to metabolic encephalopathy. Metabolic encephalopathy is capable of producing alterations in mental function, behaviour, and neurological function. This diagnosis is made due to the bizarre, rapid change of behaviour, thinking and consciousness, exhibited by the accused on the 22 August 2000. The other signs I used to base my diagnosis on were the accused's impairment of memory and orientation on the day of the incident (the amnesia).

I Disburbances of metabolism caused by hypoglycaemia can and will cause transient symptoms. Hypoglycaemia is generally associated with confusion, altered consciousness and a change in the behaviour.

(f) Hypoglycaemic Encephalopathy

A

The brain depends on glucose almost exclusively as the substrate for its oxidative metabolism. The level of blood glucose at which symptoms appear varies, and the rate of its fall may be as important as the level in determining the onset and intensity of the symptoms. An occasional patient has no signs or symptoms preceding convulsions.

(g) Convulsion

B

Convulsive seizures are common with many severe metabolic disorders especially hypoglycaemic and anoxic encephalopathies. Hence, the accused's defecation and micturition, would in my opinion be due to cerebral insufficiency giving rise to hypoglycaemic encephalopathy leading to a quiet convulsion. Due to hypoglycaemia the cerebral milieu metabolically hostile to the normal function of the cerebral cortex gave rise to psychogenic disturbances. These disturbances are manifested and characterised by varying periods of altered consciousness and behaviour leading to a twilight or 'fugue' state. The end of the 'fugue' is classically associated with the individual being unable to describe how he reached the surroundings in which he found himself in; as with the accused. It is important to bear in mind that convulsion may not be obvious. The 20 to 25 minutes of deep snoring of the accused as described by Insp T Sivakumar at the police station is consistent with a seizure.

C

D

Hypoglycaemia despite being a medical problem may present itself with psychiatric symptoms. The accused is an anxious prone personality with obsessive compulsive disorder. Consequently, he displaces his anxiety to the heart — organ fixation. Diagnostic problems faced in dealing with a hypoglycaemic patient are that it can mimic anxiety or panic attack, which also manifest in acute alcoholism. The occurrence of bizarre behaviour may draw attention away from somatic symptoms, such as it has appeared to have done so in the accused's case — 'Psycho-Somatic Over-lay'.

E

Conclusion

F

Since he became aware of what happened from the police and from his family members, his counsel, he is very chagrined and is possessed with bitter remorse.

Day by day, during the course of the trial, he is more and more saddened by what transpired in court of the alleged incident.

G

He is now suffering from Post-Traumatic Stress Disorder (PTSD) (309.81) — DSM IV (Diagnostic Statistic Manual IV) of the American Psychiatric Association in consequence of the above revelation. For this, he will need to manage his depression by taking prescription.

He has no knowledge that he had been responsible for the loss of a life. He has global amnesia of the alleged incident.

H

The alcohol that was consumed was incidental and coincidental. The blood alcohol reading is unsafe on its own and without being substantiated with clinical findings.

Be that as it may, the underlying cause of hypoglycaemia needs to be ascertained under rigorous investigation and treatment either medical or surgical soonest. Prolonged incarceration will have adverse affects on him as his anxious personality and depression will compound and it is highly likely to deepen his depression.

I

A In commenting on the characteristic features of hypoglycaemia as listed in exh D97, he said:

I have read the Article by GL Glasgow, FRACP entitled ‘The Anatomy of Automatism’ published in the New Zealand Medical Journal Vol 64 1965 at p 491. My overall clinical assessment based on the evidence in court and my personal interviews with the accused, I have found that the accused has exhibited the following characteristics:

- B**
- (i) the incident on the 22 August 2000 was offensive and bizarre. It is totally out of character with a standard of conduct of the accused in the past;
 - (ii) this abnormal behaviour of the accused appeared suddenly or explosively at about 8pm on the 22 August 2000;
 - C** (iii) there is no evidence that the incident was planned or pre-meditated. This incident was totally unexpected of the accused and especially so since the accused has never exhibited anti-social or criminal behaviour prior to this incident;
 - (iv) the accused had no motive at all and the incident was completely senseless based on the admission of the Investigating Officer. The accused exhibited confusion when he first awoke at the police station;
 - D** (v) the accused made no attempt whatsoever to conceal his criminal action;
 - (vi) the accused’s bizarre and abnormal behaviour was between 7.45–8.10pm. This period was brief; and
 - E** (vii) the accused suffers from dense amnesia surrounding his abnormal behaviour. In fact, his amnesia started from the time he was lunching with his friends at the restaurant. He does not have selective amnesia. His next memory is waking up at the police station. The evidence before the court reveals that the accused has ‘islands of partial memory’ of the events that took place after the incident. He remembers certain important events or events which are dear to his heart such as his desperate need to contact his wife, while he was at the hospital (see note P17, where his recollection of his house telephone number is perfect and yet he was confused as to the time of his arrest). He remembers writing this note to his old friend Prof Ernest Yeoh asking him to inform his wife about his arrest. He does not recall how he had new clothes to go to court the next day but he remembers having being brought to court in fresh clothes. He remembers asking for his gun on the morning of the 23 August 2000.
 - F**
 - G**

His characteristics are consistent with the criteria set down by Glasgow for his diagnosis for automatism. The accused’s state of automatism was preceded by a severe hypoglycaemia or low blood sugar attack, which had caused a transient disturbance of the accused’s brain function making the accused unaware or unconscious of his acts which led to the fatal incident on that fateful day.

H When asked on the possibility of the accused’s behaviour recurring, DW9 said:

I In the case of the accused, it is a hypoglycaemic attack. The behaviour of the accused in shooting and generally behaving bizarre has no possibility of it recurring to the extent as happened in this case. This is because it would not be possible to simulate the constellation of factors that gave rise to this severe hypoglycaemia and the bizarre behaviour. It is also difficult to have the same

social circumstances as happened here like the food, the mood and the hormone that brought about this behaviour on that day.

A

Q: Can the accused suffer recurrence of this rage attack, in any form, as a result of hypoglycaemia?

A: Everything is possible but in my opinion it is unlikely because of the long period from 1990–2001 that he has been not having any ostensible hypoglycaemia attacks. This was the first and only isolated one. Therefore it is unlikely to recur. The element of constellation of factors is relevant.

B

Q: On the assumption that the accused had an hypoglycaemia attack on that day would proper control of his diet and regular treatment ensure non-recurrence of such behaviour?

A: Yes. More importantly we must take vigorous steps why this attack occurred and eliminate it.

C

The cross-examination of DW9 commenced with his evidence on the possibility of a recurrence of the accused's behaviour. It is as follows:

It is unlikely that the accused will go through the same behaviour because of the ten-year period that he had this only once. Under the circumstances in this case it is highly unlikely that it will recur provided he is investigated and adheres to prescribed medication.

D

Q: The accused was advised in 1991 to take glucose?

A: Yes.

Q: But yet this event occurred?

E

A: Yes.

Q: How sure are you that it will not happen?

A: There is always a margin of error of 20%. Nobody in this world can be 100% certain of anything. Always there is margin of 20% error. In this case I am 100% certain because of the long interval and this is the only isolated incident and that he will not drive vehicles, use guns or ammunition.

F

Q: Supposing he does not follow this advice?

A: It is still not likely that there will be a occurrence because of his long history. The same neurophysiological constellation of factors will not operate. Constellation of factors mean the food taken on that day, the mood and the neurophysiological milieu that maintain the homeostasis of the metabolism of glucose that takes place at the cerebral cortex gave rise to the metabolic encephalopathy.

G

Q: It sounds as if the same situation as happened on the 22nd will not repeat itself.

A: Yes.

H

Q: If in future the accused does not comply with advice is it likely that he will get hypoglycaemia?

A: In my opinion, it is unlikely. For ten years he was following the doctor's advice.

Q: How did the other episodes ie the second onwards happen if he had been following medical advice?

I

A: On the other occasions he did not have enough glucose.

- A** Q: How do you know?
 A: He did not have enough glucose to the brain on that day.
 Q: How did you come to that finding?
 A: When there is not enough glucose it happens.
 I am 100% sure that the accused had hypoglycaemia on the 22nd.
- B** DW9 was then cross-examined on the counter-regulatory system in the body that supplies sugar when the sugar level in the body falls. It runs as follows:
- C** I agree that when a person is in a state of hypoglycaemia his sugar level will keep dropping unless treated. But the neuroregulatory mechanism will come into play and correct the situation in a normal healthy person.
 Q: So in a normal person before he reaches a certain stage he must have symptoms?
 A: They may have symptoms. Some will have and some will not have without symptoms. They can sweat, be confused, disorientated, stumble, lose their bearing, become aggressive, be obscene or pass out.
- D** Q: To pass out how severe should his case be?
 A: It depends on how quickly the sugar level falls and how quickly the body mechanism supplies the sugar. Passing out is getting light-headed or fainting.
- E** Q: Can a person in that state be awakened by a kick?
 A: In the Awana incident, the accused did not get up despite the kick. The accused was revived with a drink and sweets. At the same time, the neurophysiological mechanisms were in operation simultaneously. Nature was taking its course. Recovering is improvement. The accused's counter regulatory mechanism was working. It was in normal condition. It will work simultaneously with onset of hypoglycaemia. It varies from individual to individual. Usually a person has 4.0 to 5.3 millimoles of blood glucose level. It depends on each individual. If the level falls to 3.5 or lower, depending on the individual, he will show early sign of hypoglycaemia like sweating etc. The initial hormones are released. Glycogen that is stored in the liver will be converted into glucose. That will bring the level of glucose to the requirement of the individual. The person then becomes normal. There are cases where glucose level keeps on decreasing. The counter-regulatory system begins to break up and bring it back to the optimum level. If the glycogen is depleted other hormones will come into play and the central nervous system will send messages to the pancreas to stop producing more insulin. If that also fails the glucagon will be released. If that fails again the second line of defence of epinephrine starts. Even if this fails then the growth hormones comes into play. Cortisol will be released. If that finally fails it goes to the steroids. For each of this to happen, there may or may not be any symptoms. Initially there will be symptoms of sweating, confusion, disorientation. They may become aggressive, obscene. As they improve these symptoms will wane. Sometimes symptoms may not be there. It depends on individuals. After recovery, a person may not have symptoms.
- F** The clinical picture of hypoglycaemia is extremely variable. If a person did not have any symptoms he will not have any symptoms when he recovers.
- G**
- H**
- I**

With regard to the counter regulatory system to be effective it depends on the individual. No two individuals will react to a hypoglycaemia episode in the same way. The same person may not manifest the same symptoms and signs at another time at another level.

A

DW9 said that in the previous episodes the accused recovered dramatically upon glucose being given. In this regard, he said:

B

Relief by glucose is conclusive of neuroglycopenia.

The learned deputy public prosecutor then referred DW9 to DW3's book entitled *Hypoglycaemia* which says at p 413:

Measurement of blood glucose is a sine qua non for the diagnosis of hypoglycaemia. Relief of symptoms by oral or intravenous glucose, though suggestive, is not proof, of their neuroglycopenic origin.

C

In response, DW9 said:

That is his opinion (witness wants to read p 413 — witness reads). When a patient is presenting clinical features suggestive of hypoglycaemia and at that time if glucose is given and the signs and symptoms ameliorate then there is clinical proof that the person is suffering from hypoglycaemia. Dr Ridzwan did a blood test. Dr Ridzwan gave glucose. That is clinical. There is also history of treatment at the out-patient the previous day. Dr Ridzwan had done a clinical finding. He has put a provisional finding. That is normal as we are not God. He has to do further investigations to confirm the tentative finding. In this case he has not done it. If glucose is given and there is recovery and there is recurrence as in this case further investigation must be done to establish the cause.

D**E**

DW9 was then referred to p 4 of exh D85 which gave different levels in respect of the normal blood level. As he said:

Here the normal range is stated to be 3.9–6.1. Each laboratory has its own range. I agree with this range (witness referred to p 6). In this laboratory, the normal is 3.3 to 6.1 (witness referred to p 9). Here the normal range is 3.9 to 6.1.

F

With regard to tests that could have been done to establish the cause of the accused's hypoglycaemia, DW9 said:

G

I told the lawyers the hypoglycaemia condition of the accused requires serious medical attention. I have told lawyers to do the needful to allay the accused's anxiety about his imminent heart attack.

Q: Did you tell the lawyers to arrange for accused to undergo tests to find out the cause of the hypoglycaemia?

H

A: I have requested the lawyers to do that.

Q: Have any tests been done on the accused?

A: Nothing has come out in evidence that he has undergone tests.

Q: Are these tests very important?

A: Yes.

I

Q: Since it is so important, this matter should have been looked into?

A: Yes.

- A** I am not allowed to take my own apparatus to prison to check the accused. The fact about me telling the lawyers to have accused examined is mentioned in p 9 of my report. The lawyers told me that only the prosecution can initiate steps to establish the cause of the hypoglycaemia or to do any further investigation. Everything is done through the counsel. I told the lawyers to have accused referred to hospital to have his cause of hypoglycaemia ascertained. I did not pursue this matter any further. I did not tell the lawyer to have the accused to refer to the endocrine expert. I have 41 years experience in this field. Now more facilities are available. I agree it is very important to find out the cause of the hypoglycaemia.
- B**

DW9 said that when the accused saw PW8 his hypoglycaemia was at the sub-acute stage. When he was referred to DW3's views on the signs and symptoms of acute and sub-acute hypoglycaemia, DW9 said:

- C** Prof Marks is entitled to his own opinion. I have no comments. I am perfectly satisfied with my views. I prefer my views. So I say that a person in sub-acute neuroglycopenia can exhibit signs and symptoms of drunkenness. As far as research is concerned Prof Marks is world renowned. But he has been dealing only with diabetics. He is not a clinician.
- D**

DW9 said that at the time of the incident the accused was in a stage of acute neuroglycopenia. So was he when he slept at the police station. DW9 was then questioned on the convulsion that the accused underwent. It runs as follows:

- E** Q: Is there any direct evidence that accused underwent convulsion?
A: He went into deep slumber, snored loudly and after 20–25 minutes he had an obnoxious odour. I take that to be evidence of convulsion in view of his history. Convulsion is a fit and a fit can be silent. It might have been for 5–10 minutes. It was a mild convulsion or petit mal.
- F** Q: Doesn't a petit mal last for only a few seconds with full recovery and no confusion?
A: I do not agree. It can happen that way or need not. It all depends on the EEG as there are many types of convulsion. During convulsion glycogen is converted into glucose and replenishes the glucose level gradually.
Q: That is not correct.
A: I am correct.
- G** Q: Since there was no EEG you cannot be sure if there was convulsion.
A: EEG is only to tell what type of convulsion it was. There are many types of convulsion. When a person snores loudly and then smells one can from this say he had convulsion. If it is alcohol intoxication at the 4th stage, a person can go through convulsion. There are many causes of incontinence. There are many types of incontinence. I agree there is stress incontinence, urge incontinence, overflow incontinence, functional incontinence. They can be divided into two. They are persistent and acute incontinence. The accused drank beer from 2 to 7pm. He went to toilet. When the accused went to toilet, he was conscious. At the Balai, accused went into slumber. If he defecated and urinated because of alcohol, he must have been dead drunk.
- H**
- I**

He said that a hypoglycaemic patient can do complex activities. In the case of a person who is intoxicated, it depends on the degree of intoxication and

on the individual. The cross-examination of DW9 of the amnesia of the accused is as follows: **A**

When I mentioned amnesia, I mean what he could not remember on 22 August 2000. His fugue stage was from 7pm to morning of the 23rd. He had amnesia because he had no recollection of the whole incident. He doesn't remember the whole incident. He told me this when I met him in the prison. What I mean is the amnesia preceded the event. What I mean is his subsequent failure to recall. The period he cannot recall are after lunch till the next morning. The amnesia is because of his severe hypoglycaemic state. It is because of the hypoglycaemic episode which started around 7pm. **B**

Q: If that is the case he should remember what happened at the drinking session. **C**

A: He does not remember. So he tells me. He remembers the breakfast his wife prepared for him. He can also remember that his secretary forgot to inform him of the meeting. He remembers the meeting and the lunch.

Q: Is it not strange that he cannot remember the period from 3pm to 7pm?

A: He does not remember after the drinks and lunch. He does not remember leaving the restaurant. **D**

Q: If the hypoglycaemic state started at 7pm why is it he cannot remember from 3pm?

A: He must be going into hypoglycaemic unawareness at about 3pm-4pm.

Q: If he was going through the hypoglycaemic unawareness at that time at what stage of hypoglycaemia was he in at that time? **E**

A: It is difficult to know whether it was acute or sub-acute stage but he was going through hypoglycaemic state.

Q: He cannot be at that acute stage as he would have had the symptoms at that stage?

A: The clinical picture of hypoglycaemia is extremely variable. He may sweat. He can talk confused. He can become disorientated. He can lose his bearing. He can become aggressive, obscene or pass out. **F**

Q: If he was in sub-acute state he would still be conscious?

A: Once he is in the hypoglycaemic state his consciousness is impaired and there is a temporary disfunction of the mind. I maintain that once a person is in a hypoglycaemic state his consciousness is impaired. There are different levels of impaired consciousness. They are clouded consciousness, delirium, stupor and coma. At 3pm to 4pm he was in the altered state as he could not remember. Altered state is a state of automatism. **G**

Court: What is the meaning of automatism?

A: A person who carried out complex activities in a state of impaired consciousness is suffering from automatism. In an altered state, there is no full awareness. In a clouded stage he may be partially aware. **H**

(DPP reads from p 91 of Dr Marks' book under sub-acute neuroglycopenia)

Q: What do you mean when he says consciousness is retained?

A: He is referring to people who are fasting and are going into hypoglycaemia. The question of consciousness being retained does not apply to the accused as Vincent Marks has based his writing on his own statistical analysis. The lunch ended at about 3.00 pm. **I**

- A** Q: Can the accused's hypoglycaemia happen between 3.00 to 4.00 pm.
 A: It varies from person to person. The clinical picture is variable. Usually the digestion takes about three hours.
 Q: I say that between 3pm–4pm the accused could not be in hypoglycaemia?
 A: I do not agree.
- B** Q: The amnesia that he claimed he had is feigned and is an act of malingering?
 A: I do not agree.
 Q: There was no fugue in this case?
 A: I have followed Glasgow's seven criteria. I am perfectly satisfied from my examination of the accused that he was not malingering. His amnesia is dense or global. I used my interview techniques to ascertain it.
- C**

In his re-examination, DW9 said that a person who has been taking glucose as advised could still get hypoglycaemia if he had missed his glucose or the food that he had consumed was not sufficient or because of the humoral factors or any stress. He said that when he said that the lawyers had advised him that the accused can be examined only by the prosecution he meant it to refer to treatment purposes. Accordingly, he said, what he said in cross-examination is a mis-statement on his part. With regard to facilities for treating a prisoner in the prison, he said:

- E** As a private practitioner, I was not given any opportunity to examine a patient medically in prison. I cannot even take a stethoscope in. Such investigations can be carried out only by the prison authorities.

The last witness called by the defence was Mohd Hussein bin Mastan ('DW10'), a Deputy Superintendent of Prisons. He tendered in evidence exhs D77, D77A and B and D78. He said that the weight of the accused on 30 August 2000 was 75kg. With regard to private medical treatment that an accused person is entitled to receive in prison, he said:

- F** Peraturan Penjara 2000 s 107 membenarkan seorang OKT mendapat penasihat persendirian jumpa OKT.
- G** The defence then applied to recall PW4 and the accused. The prosecution did not object. The application was allowed. PW4 was asked to clarify what she had said earlier regarding the accused's visits to the clinic of DW6. As she said:

- H** I am aware of the visits of my husband to Dr Thiagi. (Witness referred to her earlier re-examination where she said, 'I am not aware of my husband's visits to Dr Thiagi.') The question that was posed to me the last time was whether I was aware of all the visits to Dr Thiagi. My answer was I was not aware of all the visits. I am aware of some visits of my husband to Dr Thiagi. I remember the visits to Pantai were in October 1991. I cannot really remember the date but I remember taking my husband to see Dr Thiagi for a complaint of faintish feeling. It could be before the Pantai incident. I remember calling Dr Thiagi before the visit. Kenny was not feeling good. He had faintish feeling and some palpitation. I called Dr Thiagi who asked me to give Kenny a sweet drink. I gave him a cup of milo. The two of us then went to see Dr Thiagi. I
- I**

was in Dr Thiagi's room. I cannot remember what transpired there. On that visit he did not advise Kenny to take glucose.

A

When cross-examined, she said:

I am aware of some visits to Dr Thiagi's clinic. During the first visit, I cannot recall what transpired at the clinic. When we were at the clinic the first time, Kenny was not so bad. I cannot recall what examination Dr Thiagi did. But there must have been some examination. Dr Thiagi did not tell me the cause of Kenny's feeling. I did not ask Dr Thiagi why he asked for a sweet drink to be given. I know that Dr Thiagi is a GP for Kenny. That morning, Kenny asked me to call Dr Thiagi. I cannot remember the date. I cannot recall whether it was before we saw Dr Ridzwan. I cannot recall my second visit to Dr Thiagi.

B

C

The accused, upon being recalled, said:

I visited Dr Thiagi in October 1991. But I cannot be certain of the date. It was before the visit to Dr Ridzwan. I had feeling of grogginess and palpitation of the heart. I was in the house. I went to see Dr Thiagi with my wife. I asked my wife to call Dr Thiagi first to inquire as to what was wrong with me. I ended up in his clinic. I can't remember whether my wife gave me a drink before going to the clinic. Dr Thiagi suggested I see a heart specialist. When I went to see him I did not appear that sick to him. The idea of seeing Dr Ridzwan was not discussed then.

D

I cannot remember visiting Dr Thiagi on the date 12 November 1998. But in 1998 I would have visited him for a similar complaint. I cannot remember whether my wife was with me. My complaint was also grogginess and palpitation. Dr Thiagi would have suggested that I see a heart specialist. I could have called Dr Ridzwan. But I do not think I made an appointment to see him. I have spoken to Dr Ridzwan on several occasions pertaining to my heart complaint. If I had seen Dr Thiagi alone in 1998 I need not necessarily have told my wife. May be I told her. May be not.

E

F

On being cross-examined, he said:

From the date of my arrest till now, there is not much change in my weight. On the first occasion, I met Dr Thiagi, I cannot recall the time. I cannot recall at what time I woke up. On that morning, I do not know why I became groggy. That was not the first time I had that feeling. It came off and on. That was the first time I saw a doctor about my grogginess. I am not sure whether I had that sort of feeling before that. On that visit, Dr Thiagi asked me to see a heart doctor. The second occasion I saw Dr Thiagi was in 1998. Dr Ridzwan advised me to take glucose. On the first occasion, Dr Thiagi said there was something wrong with my heart. He told me to see a heart specialist.

G

Dr Ridzwan is convinced I have no heart problem. He told me to take the glucose everyday. Dr Thiagi is a GP. I disagree that my evidence is not true.

H

When the defence closed its case, the prosecution applied to call evidence in rebuttal on the issue of medical evidence adduced by the accused. The defence raised strong objections to the application. Having listened to the submission of both parties, I ruled that the prosecution is entitled to call evidence in rebuttal and that the defence may, if it so desires, call evidence in surrebuttal. The judgment that I wrote on the application made has been

I

A reported in [2002] 2 MLJ 683 and thus, it is unnecessary for me to repeat it here. The prosecution called two witnesses. They are Dr Norshinah bte Kamaruddin ('PW18') and Dato' Dr Abdul Aziz bin Abdullah ('PW19').

B PW18 obtained her basic medical degree from the Monash University in 1985 and her MRCP in 1992. She was the Senior Registrar in Endocrinology in Universiti Kebangsaan Malaysia from 1992 to 1994. She took up clinical fellowship in endocrinology in Tokyo between January and May 1994 and a research fellowship in the same field at St Vincent's Hospital, Melbourne from 1995 to 1998. From October 1998 to September 2001 she was the consultant physician and endocrinologist at the General Hospital, Kuala Lumpur. Since September 2001, she has been the head of the Department of Medicine in Putrajaya Hospital. In that capacity, she is the senior consultant physician and endocrinologist.

C She has been following the progress of this trial and has attended some of the court sessions. She has read all the notes of evidence given to her and is quite aware of the facts of the case. She is thus aware of the accused's medical background. She started her evidence by explaining the meaning of hypoglycaemia. She said that the standard reference range for a normal blood glucose is from 3.5 to 6.0 millimoles per litre. In dealing with the counter regulatory mechanism in the body to produce glucose she said,

D The body has an extremely efficient counter regulatory mechanism which is initiated with the sole objective of keeping the brain with an adequate supply of glucose. From a level of 4.6 millimoles blood glucose and below the body initiates its counter regulatory mechanism by suppressing the secretion of insulin from the pancreas thereby being able to reduce the impact of insulin. As the blood glucose falls further the body will trigger to release of certain hormones such as glucagon, growth hormone, adrenalin and nonadrenalin at a level of blood glucose around 3.7 millimoles. Many studies have shown that the autonomic symptoms will appear at a level of about 3.3 millimoles and below. The other hormone called serum cortisol will be triggered if the blood glucose falls further. The other counter regulatory mechanism to keep the blood glucose normal includes the liver production of glucose which is called gluconeogenesis and the breakdown of glycogen to release glucose into the blood. This is called glycogenolysis. Of course in the very prolonged fasting state where there has not been any food intake, there is a further attempt by the body to supply energy to the brain at the expense of the rest of the body. This happens by the gradual breakdown of fats and muscle tissues to release fatty acids which can supply some energy to the brain. I say the system is efficient as the physiology of the human body is such that the most important component of the body is the brain. The glucose is the only source of energy which the brain can accept. Therefore, the components which maintain glucose balance is extremely efficient and involve the interplay between different organs and many hormones to keep blood glucose within the normal range. Therefore, it is extremely rare to have clinically significant hypoglycaemia in otherwise healthy individuals. Healthy individuals mean that the person is not malnourished, his organs are functioning well ie good liver and renal function, his adrenal glands are intact and he is not in a prolonged fasting state.

E The body gets its supply of glucose from food predominantly carbohydrate. This is from regular meals. The other sources are the liver which gets more

activated overnight when the person is fasting overnight. The third source is glycogenolysis which is the breakdown of glycogen into glucose. The other indirect control of glucose is suppression or inhibition of insulin from the pancreas. Food is able to provide glucose for 6–7 hours. Once a person eats a meal he should have a glucose supply for 6–7 hours. Ordinarily a person will have another meal after that unless he is fasting. Otherwise his counter-regulatory mechanism will be initiated. It is very hard to say how long this mechanism will function as it varies from person to person. Looking at the history of survivors of earthquakes and collapsed buildings who survived 3–7 days prior to the rescue we can infer that many individuals have very efficient counter regulatory mechanism to sustain survival in particular sustenance of the brain which can only utilize glucose in order to function. Scientific research studies involving the widely accepted glucose clamp technique have not performed studies on counter regulatory systems of healthy volunteers beyond 24 hours because that would be unethical. We have received many patients with severe hypoglycaemia with symptoms manifesting a seizure or a state of unconsciousness at home brought to the hospital and confirmed hypoglycaemia of blood glucose of less than 2.0 millimoles who will not recover without external assistance like intravenous glucose. For those who were brought in late and who are unconscious or comatose despite IV administration of glucose a few of them never recovered. They become a vegetative state or totally paralysed. A few have resulted in death.

A

B

C

D

The counter regulatory response is initiated when the blood glucose is well within the normal range thus avoiding any further precipitous fall in blood glucose and thus avoiding hypoglycaemia. This failure of the counter regulatory system rarely occurs in healthy individuals. It is more commonly described in Type I diabetes mellitus patients. And even in such patients the counter regulatory failure is not common. Also the counter regulatory system fails if the relevant hormones are deficient, for example, in liver failure and kidney failure and adrenal failure. The result is the body is unable to tackle the fall of blood glucose and the mechanism such as glycogenolysis which takes place in the liver will fail. When the counter regulatory system begins to function which can be as high as a blood glucose of 4.6 the patient may not experience symptoms of hypoglycaemia yet. There will be a continual process of this counter regulatory response if the blood glucose continues to fall. He may experience the autonomic symptoms at around 3.7 millimoles per litre.

E

F

G

There will be no drop in blood glucose immediately after a meal. On the contrary, blood glucose will naturally rise by about 2 millimoles per litre. Even in healthy persons immediately after a meal. The blood glucose will gradually fall to the normal glucose level within the next two hours. This pattern has been well shown in all normal persons being given the standard 75g oral glucose loading test. And beyond the two-hour following the meal the blood glucose will stabilise. The exceptions will be for patients with diabetes mellitus where the blood glucose rises higher after a meal and will remain high for hours after that and for normal persons undergoing a prolonged fasting state or in a rare situation of administration of insulin or excess production of insulin due to insulinoma. In reactive hypoglycaemia it may occur for those who had previously undergone gastric surgery and rarely ever occurs in a normal person. By 'transient' manner I mean it can occur up to four hours after a meal. However, the hypoglycaemia may be rectified by the counter regulatory system.

H

I

A When asked whether a person who has had lunch at 3pm can experience a hypoglycaemic condition, she said that the answer is in the negative in the case of a healthy person with an intact system. In testifying on the health condition of the accused, she said:

B In this case, based on the notes of evidence and the laboratory results made available to me, the accused is healthy because his height is 1.82m, his weight was 75.5kg on 10 February 2002 as noted by Dr Ridzwan Bakar, his last liver function test was normal on five occasions between the 20 November 1990, 12 June 1989, 25 October 1995 and 27 September 1999. His kidney function test was normal. Ultrasound was documented in February 2000 as normal. His chest X-ray was also reported as normal. His blood glucose was documented on four times as 5.5, 5.7, 5.6 and 1 fasting glucose of 5.6. A 3.7

C reading was documented by Dr Ridzwan in October 1991. That was random blood test. This is based on D85 (witness referred to D91). I have looked through this. I observed that on three occasions the accused had been diagnosed as having heart burn. On one of the notes dated 12 November 1998, it was written as hypoglycaemia. However, as a clinician it is inappropriate to put a diagnosis without supporting evidence of history or symptoms or physical examination or further tests to support the diagnosis. In

D this particular note there was none of the three except for the word 'syncope' which is a non-specific symptom. And with the additional note by Dr Thiagi that the patient was advised to see Dr Ridzwan. Syncope can be due to many causes such as rhythm disturbances of the heart, low blood pressure, volume deficient, valve disease of the heart and, less commonly, hypoglycaemia. I find

E the note quite confusing. It was only documented as hypoglycaemia leading to syncope and advised to see Dr Ridzwan. As a physician I find this statement rather confusing as there was no further documentation of blood pressure, pulse rate, appearance of patient or, at the very least, an ECG or a finger-print testing of the blood glucose.

(Saksi dirujuk kepada D85)

F With regard to D85 my observation is that between 1991 and February 2000 showed that Dr Ridzwan had mainly concentrated on investigation along the cardiac cause of the patient's symptoms. He had performed three treadmill tests and had done a series of blood investigations. The blood tests indicate that the patient had normal haemoglobin, normal total protein, and albumin which reflects that he is not malnourished. In other words he is in good

G nutritional health. His height and weight indicated he is of good build. A full physical examination done in 1991 was normal. The electrolyses, that is sodium and potassium results were all normal indicating that the kidney and the adrenal functions are normal. The liver function test as shown by the ALT, AST and ALP were normal. The significance is that the accused was in a good state of health. His thyroid function was normal on two occasions. The

H indicator of a gastro intestinal malignancy which is called CEA were normal on two occasions. One of treadmill test was done up to Stage Four. The last one done in February 2000 indicated that he had a good heart rate and blood pressure response following exercise indicating that he is quite fit. The only suspicion that Dr Ridzwan indicated that accused had hypoglycaemia was in 1991 when he gave a provisional diagnosis and the Random Blood Sugar was 3.9. However the Standard Reference Range for normal blood glucose is 3.5

I and above. Therefore, in my opinion the reading of 3.9 would not be a definition of hypoglycaemia. Dr Ridzwan is basically trained as a cardiologist. His subsequent investigations did not warrant a more detailed investigation

for the clinical suspicion of hypoglycaemia. If, as a physician, he had considered that level to be significant any physician of his standing would have referred the patient for further assessment in this line. Instead he had focussed mainly on cardiac assessment on subsequent check-ups. In addition to that he had prescribed Novaluzid which I am made to understand is for heartburn. If a clinician quotes a provisional diagnosis it is not a final diagnosis. It is a presumption based on a clinical suspicion only and usually the clinician would go further to confirm the final diagnosis or to refute the provisional diagnosis. Rightly Dr Ridzwan had sent for further blood tests. Based on D85 and D91 my view is that the accused is in a state of good health based on normal full blood counts, normal urine examination, normal kidney function, normal liver function on five occasions, normal blood glucose level on at least five occasions and a physical examination which did not indicate that he had liver failure. A person with liver failure will have striking features like jaundice, tremors, spider nevi which are typical red spots on the skin, distension of the abdomen, fluid can be felt within the abdomen and there may be swelling of the lower limb. None of these features were documented in either of the two doctor's reports.

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CEA is one of the measurable indicators which is able to assist us in screening cancers of the gastro intestinal system. It is useful to have as a screening method. In the case of the accused there was no evidence of malignancy. An otherwise healthy person is a person with an intact system. The systems of gastritis are quite common and can mimic those of angina of miocardinal ischemia, gastro eusophegal reflux. The symptoms indicated by accused are non-specific. From the endocrinological point of view there is no indication that the accused had any underlying endocrine or metabolic disorder.

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In commenting on the blood sugar level of the accused at the initial stages, she said:

The random blood glucose of the accused of 3.9 does not fall within the definition of hypoglycaemia. It is an accepted normal level. Following Dr Ridzwan's action plan blood tests sent subsequently showed that the accused's blood glucose was normal and because of that I believe Ridwan did not further consider hypoglycaemia as a final diagnosis or as a problem.

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In testifying on a state of unconsciousness from hypoglycaemia and a person's ability to recover spontaneously from such a condition, she said:

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A state of unconsciousness from hypoglycaemia happens at level of much lower than 2.0 millimoles, usually about 1.5 to 1.0 millimoles and the patient will not recover spontaneously without a rapid administration of glucose. If a person has that condition the counter regulatory response would have failed if it had failed at a higher level or it was countered by a disfunctional liver which is unable to compensate through glycogenolysis or through the failure of gluconeogenesis. What I mean is that the counter regulatory response would have been triggered before the patient becomes hypoglycaemic. Therefore most cases of severe hypoglycaemia are those related to insulin treated diabetes patients or diabetic patients on oral tablets. This is predominantly related to the drug administration and counter regulatory response failure would occur in a small number of diabetic patients of long standing duration. In other words it is the external administration of hypoglycaemic agents which very often lead to severe hypoglycaemia particularly in the elderly with a compromised renal function.

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- A** If the patient is unconscious or comatose solely due to hypoglycaemia he will not recover spontaneously. It is considered a medical emergency. He will require a rapid intravenous glucose or intramuscular glucagon administration. This will lead to a rapid and full recovery of the person assuming that his brain was not severely damaged from a prolonged hypoglycaemia.
- B** PW18 was then examined on the consistency of the accused's evidence with hypoglycaemia. In answer to questions, she said:
- Q: The accused was seen to be sleeping in the police station. Could it be hypoglycaemia?
- C** A: From what I read I cannot be certain whether he was sleeping or unconscious. However, he was able to wake up within an hour later spontaneously and able to converse and he was apparently coherent enough to be understood or to give instructions. If a person had been severely hypoglycaemic leading to unconsciousness he would not wake up without assistance.
- D** Q: There was evidence that accused had defecated in his pants. What could have caused it based on the facts and the evidence?
- A: There is no strong evidence towards a seizure because there was no witness and there was no suggestion that the accused had bitten his tongue which is a common occurrence in a seizure. The defecation in the pants is not specific at all and I would consider other metabolic problems he had at the time which is the alcoholic intoxication. I am stating this based on the blood alcohol reading of 198g. The other supporting evidence which makes the alcoholic intoxication likely is that the accused was able to wake up on his own and have a conversation and remained conscious even at the hospital. The behaviour of the accused is not inconsistent with alcohol intoxication when he was described as aggressive and yet coherent and able to give some instructions to contact some people he knew and was able to write on a piece of paper legibly which would not be in keeping with a person who is severely hypoglycaemic.
- E** Q: Is amnesia a feature of hypoglycaemia?
- A: Yes, it can be.
- F** Q: In what instance?
- A: That would occur in the neuroglycopenic stage affecting the brain and therefore memory. It occurs in severe hypoglycaemia.
- G** Q: In this case, the accused had lunch at about 3pm. Thereafter he cannot remember what happened after that. Can this be consistent with hypoglycaemia?
- A: No. The reason being that it is impossible for neuroglycopenic feature to manifest so soon after a meal.
- H** Q: If he actually had amnesia from 3.00 pm onwards and it was the cause of hypoglycaemia, what would have been the effect on him?
- A: The sequence of events would be that he would progress to the inability to carry out complex or coordinated task. He would be incoherent in his speech and movements. He would be confused and drowsy and progress to coma several hours later.
- I**

Q: In this case, the accused went to the toilet, paid his food bill, went through the toll, drove his car, made a call to his wife. Is that consistent with someone having severe hypoglycaemia? **A**

A: No, it will not be consistent with a severe hypoglycaemia because at such a stage of hypoglycaemia even if one were to drive one would not be able to coordinate well and would be knocking into nearby objects. There was no such evidence in this case. His car was alright when he was arrested. **B**

Q: The accused stopped his car, took out his gun, stopped a motorcyclist and said good. Later stopped a Perdana and fired a shot, then walked to an army jeep and shot and responded to the public. Can a person in hypoglycaemic condition perform such acts?

A: A person with severe hypoglycaemia will not be capable of coordinating himself much less coordinating others around him and he may also be incoherent. A general act of aggressiveness would not be different hypoglycaemia from other causes. By incoherent, I mean instructions given cannot be understood. **C**

The bizarre behaviour and aggressive behaviour is well described in patients with hypoglycaemia but in the instances that were illustrated the patients were restrained and glucose was administered. Those cases were proven through blood tests rather than history and observation alone. Unlike those patients, the accused had breakfast and lunch on that day. So one cannot infer that he had hypoglycaemia based on previous cases that were described. **D**

The behaviour of the accused on that day was an isolated event. There was no previous documented aggressive behaviour before or after that day. If a person has a condition that causes hypoglycaemia affecting cognitive functions he will manifest repeated or recurrent episodes of bizarre behaviour until the cause is found and removed. **E**

If the behaviour of the accused on that day was hypoglycaemia the recovery he had will be a full recovery and he will be his normal self. That is because the hypoglycaemia would have been corrected. Thus the behaviour of the accused after he recovered in the police station and thereafter is not consistent with hypoglycaemia. So one has to consider other causes for the aggressiveness. In this instance based on the evidence of Dr Bhawani the most obvious other cause is the alcohol intoxication. That is more in keeping with the symptoms. **F**

As a clinician, in order to distinguish between intoxication and hypoglycaemia, one has to fall back on the biochemical evidence because of the overlap of the symptoms. By biochemical evidence I mean blood tests. **G**

In this case given that the accused was nutritionally healthy, he had two meals, he had very good liver and renal functions it would have been extremely unlikely for him to have had hypoglycaemia at 1.30 in the morning. It is even less likely that he had hypoglycaemia at 7pm because he was not in a fasting state. There is no strong evidence to suggest that accused had hypoglycaemia at about 7pm. So one has to find other causes for his behaviour. **H**

When asked whether a person with hypoglycaemia can go into an aggressive presentation straightaway as testified by DW7 she said,

Physiologically the body goes through a fall of blood glucose but he will not straightaway become aggressive until he reaches a certain critical blood glucose level which affects his cognitive functions. Only in a person who had **I**

- A** repeated episodes of severe hypoglycaemia that such neuroglycopenia presentation can occur without the initial symptoms. Looking at the documented laboratory evidence of the accused there was no such state of repeated hypoglycaemia.

In commenting on DW7's evidence that it was possible for the accused to have had amnesia from the time he had lunch up to the time of the incident, PW18 said:

- B** Amnesia in hypoglycaemia signifies impaired cerebral function. Therefore, it is not possible for that to occur at the end of the meal at about 3m. Thereafter, it may be possible but that has to be in keeping with all the other symptoms that followed. Amnesia is not a specific symptom of hypoglycaemia. Neither is it reliable as a symptom.
- C**

When asked about DW7's evidence that a person in hypoglycaemia coma can recover naturally without being treated, PW18 said:

- D** No, it is not based on all the comatose patients that I have seen in the hospital brought in comatose. None of them would wake up unless an urgent and rapid administration of glucose or glucagon IM is given. This has always been classified as a medical emergency. Such patients in coma will have a blood glucose level of around 1.0 millimoles per litre. It is not possible for a person at that level to recover naturally. If the accused had been in a state of coma due to hypoglycaemia alone then he would not have recovered spontaneously and naturally.

- E** PW18 was questioned further on the evidence of DW7. In answer to the questions, she said:

Dr Elizabeth categorised accused as having sub-acute neuroglycopenia without symptoms. What is your response?

- F** A: In this case the opinion was given based on the assumption that accused had hypoglycaemia. Therefore I cannot comment on it. A person can undergo a hypoglycaemic episode without symptoms. That is called hypoglycaemic unawareness.

The biochemical evidence of blood tests remain the mainstay of the diagnosis of hypoglycaemia given that symptoms alone are not reliable.

- G** Q: Dr Elizabeth said that reactive hypoglycaemia would have started on the accused at 7 to 7.30pm. Do you agree?

A: I disagree that reactive hypoglycaemia occurred at that time because he had no history of past gastric surgery, there was no previous documented excessive levels of insulin and that reactive hypoglycaemia is very uncommon. Again this description is purely speculative.

- H** Q: Dr Elizabeth said that accused had sub-acute neuroglycopenia at 8pm and at 1.30am he may have had acute neuroglycopenia. Do you agree?

A: No. The reason being that acute hypoglycaemia is defined as a rapid fall of blood glucose which is usually due to an external administration of a drug or toxin. That did not happen in this case.

- I** Sub-acute hypoglycaemia refers to a gradual progressive decline of blood glucose.

Q: Dr Elizabeth said that the accused had a hypoglycaemia episode that began at 6.30 to 7pm with the blood sugar reaching its lowest point at

8pm and the gradual recovery occurring between 10 and 11pm. Can I have your opinion?

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A: The explanation in my opinion is based on assumption. Therefore, I disagree with that as a diagnosis given the facts and data shown to me indicating that on five separate occasions the laboratory blood tests of blood glucose were found to be normal. Sub-acute hypoglycaemia does occur but for the onset of acute hypoglycaemia to occur administration of certain substances would have to be introduced for that to take place. There must be an intervening factor. In this case, there was no intervening factor.

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Q: Dr Elizabeth said that the accused's behaviour suggests that the counter regulatory mechanism in his body failed to maintain the blood sugar level and that the system that ought to have worked failed. Is there any justification for this?

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A: In my opinion, there is no justification for that explanation because falling back on the available data as proved the accused has intact liver function, intact renal function, is not malnourished and the blood glucose levels previously were normal and he did not appear wasted meaning thin and lacking in muscle mass thereby concluding that his counter regulatory system is in good condition.

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I disagree that there was a failure of the liver system to correct the falling blood sugar because there is no physical or biochemical evidence of liver failure.

Q: Dr Elizabeth said that there is enough evidence to suggest that accused had hypoglycaemia and that she was positive of this even without any clinical finding. Do you agree?

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A: I disagree because as a physician and endocrinologist symptoms must always be supported by scientific data or other biochemical proof given the great overlap of symptoms pertaining to hypoglycaemia.

In commenting on the accused's loss of consciousness in Awana, PW18 said:

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With regard to the Awana incident if the accused had taken sweets before coming to the lounge it is not possible for hypoglycaemia to occur because taking sweets is the treatment of choice to correct mild hypoglycaemia leading to a full recovery. Therefore, his unconsciousness at the lounge may not be caused by hypoglycaemia. It may be of cardiac origin. I would leave the diagnosis open as there was no further investigation.

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PW18 was cross-examined on the normal blood level. As she said:

I need to add that because laboratories differ in their reference range for most tests because of the differences in the cohorts of the population. The figures given here can vary from laboratory to laboratory. The minimum range need not be 3.5. For example, in Dr Ridzwan's laboratory document the fasting range is 3.3 to 6.1. In D85, it is 3.9 to 6.1 under University Hospital. I have seen variations in reference ranges. The difference in the range is based on the species of the population. We do not rely solely on a blood glucose alone. It has to be in the context of other presentations. I am not sure if there was a study conducted to determine the median range of normality of blood glucose in the population. I am not surprised that there is none. I agree that the figures used are arbitrary. (Witness referred to D98) For 50% of the people it is 4.9. For the 45–54 age range for 50% it is 5.2. I agree for women it is different. P121 makes no distinction between males and females.

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A She agreed that there are people with a reading of 3.0 millimoles and are normal without symptoms. This is especially true of ladies. On the other hand, there are men who got the symptoms at 4.1 millimoles. In commenting on the blood level of the accused, PW18 said:

B I do not know whether the blood tests in Dr Ridwan's report are fasting blood or random. One of four readings shows it is fasting. All the laboratory tests indicate above 5.0. From 30 October 1991, the reading was 3.9. This is not an uncommon observation. For any individual there is always a range. From the notes that was the only occasion he had 3.9. For 23 October 1991 it was 71mg. It is 3.94 millimoles. The figures can be meaningful only in the light of the doctor's clinical interpretations of the symptoms of the patient. I agree Dr Ridwan is a competent physician.

C When cross-examined on the loss of consciousness of the accused at Awana, she said:

D When a person has symptoms and is given glucose he will recover from 3–10 minutes depending on the patient and depends on the type of sweets. With regard to the Awana incident, I have no idea how long the accused took to reach the clubhouse. He could continue into a worse state of hypoglycaemia. If he had arrived at the clubhouse earlier he would not have recovered. If the sweets did not take effect yet he could collapse. My evidence on this earlier was that he was conscious to take the sweets. If the sweets had not taken effect yet it is possible he could have collapsed when he reached the clubhouse.

E PW18 agreed that at the time of the incident there was something that was affecting the mind of the accused. She said that she has considered the probable causes of hypoglycaemia and intoxication. In saying that the cause could be intoxication, she said:

F I disagree that the evidence of the three witnesses at the scene did not support intoxication as a cause because the accused had been described as confused; there was a question of him being disorientated although he remained steady; he was obviously aggressive and disinhibited; he appeared agitated when his instruction was refused. This may be in keeping with alcohol intoxication although in my limited experience in studies of alcohol intoxication and falling back on my clinical experience a person who has had a chronic heavy consumption of alcohol usually has a high tolerance to the effects of it and will present with some, if not all the features, of acute alcoholism. I have not heard of Kurt Dubowski. I have read fully D84. We do not remember all pioneers in all fields as medicine is a very broad subject (witness referred to D84 p 21). From the notes given to me, the accused and his wife had described the drinking habits of the accused. That shows he was a chronic heavy drinker. Such a drinker need not have necessarily have an effect on the liver. A chronic heavy drinker may still be normal in respect of the physical signs and the laboratory findings that are made available. Prof Marks described accused as moderate to heavy drinker. The accused has a high tolerance level to alcohol. From my limited knowledge of alcohol it means that he is still able to carry on a habitual task without being affected by the alcohol. At that time, the accused may have broken down in tolerance. The high tolerance refers to what he normally does. That does not mean that he is unable to go into a rage. The accused appeared to be able to have physical control of his body and movement but his cognitive and higher mental functions and a sense of

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judgment was severely impaired (witness referred to table at p 21). The alcohol level of accused was 198mg (witness referred to 'Excitement' and 'Confusion' in table). I would put the accused in the 'Confusion' stage. However, like in many pathological cases in medicine there may be overlap of symptoms and there need not be the manifestations of all features described in books because there are usually individual variations as in all aspects of medicine. I do not have an authority to support it. It is based on my clinical experience of individual experiences that may vary. As a junior officer, I have come across it. I have been in the Accident and Emergency Department for about three months in Johore Bahru.

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When asked to comment on whether the characteristics mentioned in exh D97 had been satisfied, she said:

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The first characteristic is met in this case. The second characteristic has also been met. The third characteristic is also met except that I cannot explain the position of his car. However, I cannot say whether it was planned or not. In this regard I do not know what the author means by 'planned'. That is why I have to be very cautious. If it refers to consciously planning there is no evidence. I agree that the 4th characteristic is met. I agree that the fifth characteristic is met as accused was confused. With regard to the sixth characteristic the accused was forcibly restrained some minutes after the incident therefore curtailing any possible contention of behaviour. Therefore I cannot comment on the sixth characteristic. There was no described evidence of misbehaviour by the accused after he was arrested. I am not disputing that the incident till the arrest took about one hour. With regard to the seventh characteristic accused had periods of amnesia.

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In commenting on hypoglycaemia as a probable cause, she said:

If the accused had food at 3pm reactive hypoglycaemia may explain the behaviour five hours later but it cannot explain the subsequent behaviour of the accused from 8pm. It cannot explain what happened at the police station and hospital. It can explain the behaviour of the accused at 8pm if it was hypoglycaemia. The length of hypoglycaemia depends on the drop of blood level. Hypoglycaemia can last from 1–5 hours. It depends on the degree of hypoglycaemia. It can go into a convulsion. A convulsion can be in the form of a petit mal where there need not be the biting of the tongue. I am not qualified to agree whether petit or grand mal is nature's way of recovering. A petit mal convulsion may last between 1–10 minutes.

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The accused was snoring in the police station. Petit malconvulsion may be one of the explanations of the features of deep sleep for 20 minutes and snoring. In the case of intoxication incontinence can take place when he is deeply unconscious and not when he is near death (witness referred to D84 p 21). According to this table it will come under the 400–500mg. A reading of 198gm of alcohol is consistent with six glasses of beer bearing in mind the time it was collected. If alcohol was the cause the accused would perhaps have slept off instead of waking up and saluting. A convulsion can lead to incontinence but it is not exclusive to hypoglycaemia (witness referred to D21 pp 24–25). This may explain why accused did not fully recover but in my own experience with diabetic cases recovery is not possible without help. Very rarely there can be recovery without assistance. I am not an expert on alcoholism. I do not know why he behaved that way in the hospital and not before. I am not well versed with the absorption rate of alcohol.

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A PW18 said that in severe causes of hypoglycaemia a patient will not recover unless aided. She added that without assistance the general rule is that they will go into unconsciousness and may not recover. When challenged on this view, she said:

B I have seen cases of severe hypoglycaemia resulting in coma and death. It was death in the hospital. I can recall one case which happened two to three months ago. It is on electronic medical record in our hospital. I remember the facts of the case. In that case the patient was an elderly lady with long standing diabetes on oral hypoglycaemic agent. She was in her sixties. She developed hypoglycaemia coma. I am not sure whether she was brought in a coma. She subsequently died. Her blood reading was about 1.0 (witness referred to D82 p 80). I do not dispute the statement. The cases that I encountered were all exclusively diabetic patients taking anti-diabetic treatment. I have treated non-diabetic patients for hypoglycaemia. I cannot quantify. There have been several. Perhaps 50–100 in the course of my career of varying causes of hypoglycaemia in Malaysia. The causes I encountered are due to chronic end-stage renal failure, chronic liver disease, liver failure, Edison's disease (a state of low cortisol), hypopituitarism which results in combined or multiple hormone deficiency, insulinoma, aspirin overdose and of course the insulin treated diabetes patients. There are many more underlying causes. I have not treated reactive hypoglycaemia. I have treated alcohol induced hypoglycaemia. 10% of causes are idiopathic. I have not come across such cases. In the 50–100 cases I saw, they are related to some organ problem already existing. There are cases where patients went into hypoglycaemia two to five hours after taking food. That is reactive hypoglycaemia. It is not reacting to the food but reacting to the insulin secretion that reacted to the food. If there is literature to show that alcohol induced hypoglycaemia can come by itself I will accept it. I have read about the Gin and Tonic Hypoglycaemia. I have not read articles about it (witness shown a document). Shermin and Kerr are well known. I would like to read this before commenting on it — marked as D125.

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When asked to explain the delay in the peaking of alcohol in the accused, PW18 said:

G I have no explanation for the delay. However, the single documented reading of 198 mg did not necessarily mean that that was the peak at collection time at 1.00 am because there is no other comparative reading. I cannot comment on Dr Marks' evidence that there could be a possible hypothesis that blood lacking in sugar would impede absorption of alcohol because there have been no studies on it.

H PW18 said that the disorientation of the accused as to time and place can be a feature of coming out of hypoglycaemia or alcohol or both. When re-examined on the normal blood glucose level, she said:

I (Witness referred to D121) The figure is arbitrary because in all laboratories blood glucose readings reference levels are given as a range and not as absolute readings. This is because of the variations amongst normal individuals. There is a certain standard action plan that physicians go through when a certain blood glucose level is normal. It can be accepted as normal if within the clinical context. If the blood glucose equivocal be it on the borderline, low or high a further assessment or determination of that reading will have to be

made. Equivocal readings are not taken as a diagnosis. Everywhere there is a range that is adhered to. Most authorities will take the level of 3.9 and above as being normal. Dr Marks said that the blood glucose of 3.9 should be accepted as normal. In D126, it is stated at p 850 under the para 'Nomenclature' also as 3.9. (Witness referred to D124) At p 948 it also states 3.9 as being normal. At p 939 of D124 it is also stated that the normal range is between 3.9 to 8.3 despite wide variations. In short most authorities take 3.9 and above as being normal.

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In dealing specifically with the blood glucose level of the accused, she said:

In general, the medical history of a person is important to formulate a diagnosis. In the context of the accused's medical history the symptoms presented were not specific and they were not pathognomonic (highly specific) of hypoglycaemia. The symptoms are commonly mimicked in other conditions or stress state. Secondly, looking at the blood glucose taken from Dr Ridzwan's notes were equivocal, ie 3.9 and these were done as glucometer technique as verified by Dr Marks' testimony. We know that glucometer readings should not be accepted for diagnostic purposes because of the variability of the result depending on the end user technique and the lack of quality control of the kit as opposed to laboratory readings and performance. I am not saying that the glucometer is not useful. It is very useful for patients' self care and monitoring purposes. But it is not accepted and it is not one standard practice to use the glucometer for confirmation or diagnosis of hypoglycaemia or hyperglycaemia. Confirmation of hypoglycaemia requires blood glucose to be collected and sent to the laboratory using the proper method.

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When she was referred to exh D97, she said:

With regard to third characteristic I cannot agree because the accused had his car across the road and was instructing people to get out of their vehicles. He was acting to a certain instruction that he was giving. He was also reacting to the response given by the people he ordered. So I cannot be certain whether this means the act was planned or not. With regard to the sixth characteristic I also disagreed because, one, he was forcibly restrained, two, he continued to exhibit abnormal behaviour even after he woke up at the police station and at the hospital. So it would not fit with the sixth characteristic.

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When asked whether the accused was in a state of automatism at the material time, she said:

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Automation would fall in the sub-group of neuroglycopenia because it reflects abnormal behaviour and therefore disfunction of the brain. For that to occur blood glucose level would be expected to be in the low range of about 2.8 millimoles at its highest point based on most literature. Based on the factual situation, it is very unlikely had such low blood given that he had normal meal followed by alcohol which was customary in him and his other compensatory mechanisms are otherwise intact ie intact liver, renal function and he was not sick or ill on that day. Therefore, if hypoglycaemia were about to occur, his counter regulatory mechanism would be able to compensate each other to normalize his blood glucose. There must be a state of hypoglycaemia well below 3.0 millimoles to undergo the state of automatism.

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In expressing her view that the accused did not have hypoglycaemia, she said:

- A** As a general rule, alcohol may induce hypoglycaemia and this happens by the inhibition of gluconeogenesis but it does not inhibit glycogen breakdown to glucose. In this case assuming that accused did become hypoglycaemia alcohol may have induced hypoglycaemia in him. In my opinion, the accused did not have hypoglycaemia because he had a full meal prior to the incident, he was in good nutritional status, his physical characteristics from previous testimony had ruled out any hormone deficiency, blood tests showed intact blood count, liver and kidney functions thereby suggesting that he would have good preservation of glycogen storage which can compensate for any fall in blood glucose if there was one.
- B**

- C** PW19 graduated with a medical degree in 1974. He obtained a Masters degree in Psychological Medicine in 1981. He also obtained a Masters degree in Public Health majoring in community mental health in 1982 from the John Hopkins University in Baltimore. Upon his return, he worked as a psychiatrist and is now the head of the Department of Psychiatry, General Hospital Kuala Lumpur.

- D** PW19 had followed the progress of this trial. He had been present in court from time to time and is familiar with most of the facts of the case. He had examined the accused for about an hour on 8 October 2001 at the Sungei Buloh prison. In commenting on the results of his examination, he said:

- E** As always in any examination of a psychiatric case, more particularly a forensic case, I had introduced myself to the accused and others present. I explained the reasons for the interview and evaluation and my job as a psychiatrist. After a short formal introduction, the psychiatric interview went on very well for about an hour. In my examination of the accused, I found that his general condition to be very satisfactory. He is tall, slim and neatly attired in T-shirt and white pyjamas and well-groomed. Although he was brought in handcuffs, it was off for the whole interview. He spoke in a spontaneous and fluent English with expressions and gestures. Initially, he was a little tensed and anxious especially when talking about the event. Later, he was cheerful and apologetic. His mood was found to be eurhythmic though tearful when recalling events of the incident. There was no disturbance in mood or thought. Cognitive functions were intact. He denied any past history of psychiatric illness or any significant medical problem. In his family and personal history,
- F**
- G** he was found to be a married person, worked as a company director and admitted to taking liquor regularly for 30 years. Therefore, based on my short examination of the accused and evaluation of the case, my working diagnosis at the point of evaluation then was no significant psychiatric morbidity.

- H** PW19 tendered in evidence the report that he had prepared on the accused (exh P127). In outlining the most probable cause for the accused's behaviour at the material time, he said:

- I** Taking into account his history, examination and testimony in court at the time of the incident the most probable diagnosis was alcohol abuse in intoxication. The criteria for making such a probable diagnosis is based on diagnostic statistical manual IV: (a) history of recent ingestion of alcohol hours prior to the incident; (b) clinically significant maladaptive behaviour and psychological changes; (c) one of the six signs, ie (i) aggressive; (ii) impaired judgment; (iii) impaired social functioning which happened

shortly after the ingestion; (iv) unsteady gait; (v) nystagmus; (vi) stupor or coma; (d) no other medical condition to account for the above which I have mentioned. This is further supported by the blood alcohol level of 198 mg. It is also my opinion that it is not unusual for persons who have been accused of any homicidal offence to remember events in considerable detail but to have no memory of the actual incident i.e. amnesia. It is also my opinion that it is extremely difficult to differentiate psychogenic amnesia or motivated forgetting due to organic cause. Psychogenic amnesia means motivated forgetting or deliberately not remembering events or experiences of the past.

In saying that it is unlikely that the accused was in a fuge state, he said:

On the facts of this case, it is most unlikely that the accused was in a fuge state. When there is probable cause which can be described as contributory to the state, as in this case intoxication, fuge state is almost always excluded. In this case alcohol is important as there was alcohol consumption prior to the event and the subsequent blood reading.

There are set criteria to diagnose fuge. It is well described in DSM IV. (Witness refers to a document) It is at p 484. It does not fulfill any of the criteria described here. None of the criteria fits into this case. The criteria are: (a) the predominant disturbance is sudden, unexpected travel away from home or one's customary place of work with inability to recall one's past; (b) confusion about personal identity or assumption of new identity partially or completely; (c) the disturbance does not occur exclusively during the course of a disassociative identity disorder and is not due to a direct physiological effect of a substance for, eg drug or epilepsy; (d) symptoms caused are clinically significant distress or impairment in social, occupational or other important areas of functioning. These are the four criteria which must be fulfilled before making a diagnosis of fuge.

In commenting on the evidence of DW9, PW19 said:

I have read the evidence of Tan Sri Mahadevan and his report. I have found that the evidence has been based on several assumptions. He mentioned that the accused was definitely hypoglycaemic. I find it very difficult to agree with it. Although the report is very lengthy there are terms in psychiatry which are definitive which are loosely used by him. It is normal practice in psychiatry for our reports to be brief and concise and direct to the case in question and based on facts which we have examined. There are times I found the report somewhat confusing to comprehend. It is normal practice in psychiatry for the facts to be first considered plus a physical and mental state examination of the case. There are times we must do confirmatory tests to confirm our finding. There was a mention of snoring which is (at p 23) 'snoring was in fact a quiet convulsion (as epileptic form of attack but not actually epileptic).' This is not true. Snoring can be caused by other conditions like sleepapnoea. I do not have anything else to say on the report. Just one more thing. In psychiatry, particularly in forensic psychiatry, final diagnosis is based on facts physical and mental status examination and laboratory findings. There are specific criteria for us to consider in making a definitive diagnosis. Provisional diagnosis may be entertained but has to be supported by evidence which I find difficult to find in the report.

In his cross-examination, PW19 agreed that symptoms produced by hypoglycaemia and alcohol can be so identical that it can be confusing even to a doctor. When questioned on his examination of the accused, he said:

- A** For a psychiatric assessment, it may require more than one interview. A one-hour interview may not even be enough for a provisional diagnosis.

As he said further:

- B** The interview with accused was conducted by me. My assistant took down the notes. I have heard part of the evidence of Dr Ridzwan. I have also heard part of the evidence of Dr Bhawani. I heard a good part of the evidence of Dr Vincent Marks. I did not hear the evidence of Dr Elizabeth and Dr Norshinah. If the accused did not take his medical condition seriously it is not a surprise to me. He told me he had no past history of psychiatric illness. He said he had no significant medical problem. He was seeing Dr Mahadevan regularly. The accused had no past history of violent behaviour. He told me he had been drinking for the past 30 years. He told me he could take half a bottle of whisky a day and hold it. I would regard him as a heavy drinker. I did not find out whether accused had withdrawal symptoms in the prison. I would not be surprised if he had no such symptoms. The behaviour of the accused is most likely alcohol related going by the diagnostic material available. I have heard of the Kurt Dubowski chart (witness referred to D84 p 21). The accused must have been affected by something to have behaved like that. In this case the issue is whether it is alcohol or hypoglycaemia or a combination of both. There is evidence to show that the accused was aware of certain things. The accused was stopping traffic on the road. The accused stopped the taxi driver and then told him to go. The accused was also trying to shoot when there were no more bullets. Two witnesses said he was walking aimlessly. He was trying to stop a taxi which had no driver. The accused was confused and somewhat disoriented. There is a likelihood that the accused did not know what he was doing. If a person is so intoxicated he will not know what he is doing. I agree that in the chart the accused must have been in the category of 'confusion'. In the case of alcohol blackout it will take sometime for a person to wake up. In a petit mal convulsion a person will get up in 20 minutes or shorter. Defecation happens in alcoholic cases. Snoring is not what we look for in petit mal. It is not a feature of petit mal. There will be a short memory loss. I will not be able to say whether it was petit mal or not. I do not disagree. Petit mal will produce a vacant gaze upon waking up but not necessarily all the time. It does not involve biting of the tongue (witness referred to D84). From this table to defecate one would have to be at the last two levels. But there are other tables.

- G** In commenting on the Ganster test conducted by DW9, PW19 said:

It is difficult to distinguish between amnesia which is genuine and malingering. Ganster test is a prison psychosis. It is not a test but is part of the interview. If a person is pretending he will give approximate answers. In this case, it is not relevant as accused does not have ganster syndrome at all. There are other tests that can be conducted. I did not have the time to do it.

- H** PW19 said that he did not investigate the aspect of hypoglycaemia in this case. He also said that the Dubowski chart may not apply in the case of the accused as it varies from person to person. He agreed that a fugue state can happen in a hypoglycaemia attack. When referred to p 492 of exh D97, PW19 said:

- I** In automatism, the behaviour is instantaneous as in condition (2). It is difficult for me to agree with condition (2). Condition (4) exists. Condition

(5) exists. With regard to (6), the abnormal behaviour was for more than an hour. No (6) is not fulfilled.

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In his re-examination, PW19 said that his report is not based on his interview of the accused alone. It is also based on his presence in court. As to whether hypoglycaemia is a probable cause of the accused's behaviour, PW19 said:

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If I had the finding of Dr Ridzwan at that time my finding would not have been altered as it is only provisional. With the history of the accused and the sources available now I am still not convinced of hypoglycaemia.

He said that the Dubowski chart is not a strict test. It depends on the individual concerned, his ethnicity, his tolerance, pre-disposition and other factors. He said that the Ganser test is used for psychosis and not to determine whether a person is feigning amnesia. In commenting on the behaviour of the accused at the material time, PW19 said:

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When I said accused was aware of the surroundings I mean he was aware that he had used the gun by shooting at people; he knew what the gun is used for; he stopped traffic; he went to the taxi wanting to go home.

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(Witness referred to D97) What is abnormal of the accused is the driving; the stopping of traffic; shooting at random; at the hospital he was abusive. That was the period of the abnormal behaviour.

He concluded by saying that hypoglycaemia and alcohol are not his areas of expertise.

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The defence did not adduce any evidence in surrebuttal.

In the light of the submission of both parties at the end of the case, I shall first deal with the law relating to hypoglycaemia as a defence. It is one of the ailments that leads a person to act in an automated state. Automatism refers to a state of defective consciousness in which a person performs unwilld acts. It is related to the requirement of voluntariness and mens rea. In *Ryan v R* (1967) 121 CLR 205, Barwick CJ emphasized that the word 'automatism' is just a convenient expression for the absence of voluntary conduct. To that extent the twentieth century common law innovation harmonizes with s 39 of our Penal Code which reads as follows:

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A person is said to cause an effect 'voluntarily' when he causes it by means whereby he intended to cause it, or by means which, at the time of employing those means, he knew or had reason to believe to be likely to cause it.

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The meaning of 'automatism' was exhaustively dealt with by the House of Lords in the celebrated case of *Bratty v Attorney General for Northern Ireland* where Lord Denning said at p 409:

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The requirement that it should be a voluntary act is essential, not only in a murder case, but also in every criminal case. No act is punishable if it is done involuntarily: and an involuntary act in this context — some people nowadays prefer to speak of it as 'automatism' — means an act which is done by the muscles without any control by the mind, such as a spasm, a reflex action or a convulsion; or an act done by a person who is not conscious of what he is doing, such as an act done whilst suffering from concussion or whilst sleep-

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- A walking. The point was well put by Stephen J in 1889: ‘Can anyone doubt that a man who, though he might be perfectly sane, committed what would otherwise be a crime in a state of somnambulism, would be entitled to be acquitted? And why is this? Simply because he would not know what he was doing,’ see *Reg v Tolson* (1889), 23 QBD 168, 187. The term ‘involuntary act’ is, however, capable of wider connotations: and to prevent confusion it is to be observed that in the criminal law an act is not to be regarded as an involuntary act simply because the doer does not remember it. When a man is charged with dangerous driving, it is no defence to him to say ‘I don’t know what happened. I cannot remember a thing,’ see *Hill v Baxter* [1958] 1 QB 277. Loss of memory afterwards is never a defence in itself, so long as he was conscious at the time, see *Russell v HM Advocate* 1646 SC (J) 37; *Reg v Podola* [1960] 1 QB 325; [1959] 3 WLR 718; [1959] 3 All ER 418; 43 Cr App R 220 CCA. Nor is an act to be regarded as an involuntary act simply because the doer could not control his impulse to do it. When a man is charged with murder, and it appears that he knew what he was doing, but he could not resist, then his assertion ‘I couldn’t help myself’ is no defence in itself, see Attorney-General for *South Australia v Brown* [1960] AC 432; [1960] 2 WLR 588; [1960] 1 All ER 734, PC: though it may go towards a defence of diminished responsibility, in places where that defence is available, see *Reg v Byrne* [1960] 2 QB 396; [1960] 3 WLR 440; [1960] 3 All ER 1; 44 Cr App R 246, CCA: but it does not render his act involuntary so as to entitle him to an unqualified acquittal).
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- E In explaining the meaning of an act done by the muscles without any control of the mind in the judgment of Lord Denning, North P said in *R v Burr* [1969] NZLR 736 at pp 744–745:

- F In my opinion then there is now clear judicial authority for the view that in order for a defence of automatism to succeed, the person whose conduct is under review must be unconscious of what he was doing. In short that what he did was an unconscious involuntary act. Now, Mr Davison submitted that the categories of situations in which automatism may arise cannot be said to have been closed. That may be so but, in my opinion, the evidence must be sufficient to lay a proper foundation for the plea that the accused person acted through his body and without the assistance of his mind, in the sense that he was not able to make the necessary decisions and to determine whether or not to do the act. Now in the present case all the facts show that the appellant knew what he was doing and knew that what he was doing was wrong. This being so, in my view there was no room at all for the defence of automatism. It is not sufficient that the medical evidence suggests that the appellant’s mind was not fully functioning and that he had an imperfect appreciation of the nature and quality of his act. To allow such a plea to be submitted to a jury in a case like this would be dangerous in the highest degree. It would be quite contrary to the view the law has always adopted in the case of an insane person for he is liable unless he is able to meet the tests laid down in the McNaghten rules, which, as I have said, are now enshrined in our Crimes Act. I think it should be made plain that when Lord Denning speaks of ‘an act which is done by the muscles without any control by the mind’, he does not mean that the accused person must be absolutely unconscious because you cannot move a muscle without a direction given by the mind. What his Lordship in my
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- I opinion was saying is that all the deliberative functions of the mind must be absent so that the accused person acts automatically. Lord Denning, a little later in his judgment, said: ‘Nor is an act to be regarded as an involuntary act

simply because it is unintentional or its consequences are unforeseen' (ibid, 409, 532.)

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In *Police v Bannin* [1991] 2 NZLR 237, Fisher J adverted to the fact that in automatism the difficulty is that there are many possible levels of consciousness and, consequently, it may not be surprising that the cases have not always been easy to reconcile. As his Lordship said at pp 249–250:

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In some, mere impairment of consciousness, understanding and reasoning power has been held insufficient, eg *Broome v Perkins* (1987) 85 Cr App R 321 (it was not sufficient that the defendant on a charge of driving without due care and attention was in a hypoglycaemic state, because he still had sufficient conscious control over his car to be able to brake violently and veer away from other vehicles); *R v Isitt* (1978) RTR 211 (the accused in an hysterical fugue due to a collision which he had caused, then drove dangerously while successfully evading police; his memory had ceased to function, his subconscious mind had taken over and he was said to be 'totally unaware of things like legal restriction in terms of what he did, totally aware of moral concern in this fugue', but his driving was considered purposeful in that he wanted to get away from the accident); *Haynes v Ministry of Transport* (1988) 3 CRNZ 587 (the defendant, apparently concussed in an accident, was suspected of drinking but refused to give a blood sample; expert evidence was that at the time of refusal he was probably not fully conscious or able properly to appreciate the request or able to make a fully informed or rational decision and he was said to be 'capable of voluntary action but ... improbable he was capable of fully conscious voluntary action'; it was held that mere mental impairment was not to be equated with automatism). Yet in others, the defence has been upheld notwithstanding at least a residual degree of awareness, eg *R v K* (1971) 2 OR 401 (the accused, who killed his wife while in shock when informed she was leaving him, was described as in 'a state of diminished awareness'); *R v T* (1990) Crim LR 256 (in a state of shock after being raped three days earlier, the accused participated in an armed robbery involving an initial scuffle with the victims, leant into the victim's car and took her handbag, and was later described as 'acting as though in a dream' with only partial recollection, but note the relatively complex purposive action); *R v Price* (19 November 1949) footnoted in Williams, *Criminal Law: The General Part* (1953) at p 11 no 4 (a marine attacked a corporal with a bayonet while in a 'clouded, twilight state of consciousness'; not having fully recovered from a dream.

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In considering the proper test to be applied, his Lordship said at p 251:

A common way of approaching automatism is to define automatism itself, and then to see whether the accused falls within that preconceived definition, rather than to start with the mental elements of the crime charged and then to see whether the accused lacks the capacity to satisfy those elements in the instant case. In the finish, there should be no difference in the result. But I cannot help thinking that the debate over differing levels of consciousness, and the possibility of consciousness without voluntariness, has not been assisted by the tendency to concentrate upon automatism as though it had a life of its own. The ultimate question is not whether the case falls within a preconceived definition of automatism or is mentally incapacitated to some preconceived extent. There is little point in addressing mental states in the abstract. Automatism as a concept is no better than a negative and indirect route to the

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- A** question that really matters. The question is whether the accused has retained sufficient mental capacity to reach the threshold required for the particular crime with which he is charged. That threshold will vary from one type of crime to another. It will be affected by the reasons for which a particular class of conduct was proscribed by the law, the implied statutory intention as to the level at which citizens should be held responsible for their conduct, and the way in which the law has defined the crime. In short, the focus should lie upon the elements of the charge.
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As his Lordship concluded at p 250:

- C** I think that while ‘unconscious involuntary action’ will suffice as the test for obvious automatism cases, something more sophisticated will be needed for marginal ones. At least in New Zealand, it has been recognized that total unconsciousness is not necessary for the defence. It is sufficient if ‘all the deliberative functions of the mind (are) absent’ per North P in *R v Burr* [1969] NZLR 736 at p 745.

In dealing with what constitutes the deliberative functions of the mind, his Lordship said at p 254:

- D** To summarize, the defence of automatism can be reduced to the question whether at the material time the accused had the mental capacity to form the particular mental ingredients of the crime with which he is charged. For this purpose, no distinction is to be drawn between sane and insane automatism. That distinction affects only the onus of proof and the ultimate disposition of the case. The mental ingredients of the crime will vary from one case to another but in every case the accused must have: (i) some appreciation of each of the key facts relevant to the crime; (ii) some capacity to make a decision to act with respect to those facts; and (iii) some capacity to form each of the residual mental elements of the particular mens rea involved. It will be no defence that those capacities might be severely impaired, that normal inhibitions might be absent or that there might be a compulsive urge to act in that way.
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His Lordship then explained the manner of determining whether the deliberative functions of the mind are present at p 253:

- G** It follows that awareness of the relevant facts to any degree qualify as the first of the three capacities I have suggested, however clouded, confused and distant that awareness might have been. In that respect, one of the obvious tests is that of memory. Although subsequent recollection is not essential to awareness at the time, it is difficult to see how there could be recollection later without prior awareness to the standard required. Nor do I think that in itself patchiness in the scope of periods of awareness should matter. What matters is whether the accused was aware of each of the particular facts essential to the crime. A similar approach seems appropriate to the other capacities to which I have referred. The fact that reasoning powers are much impaired does not of itself negate contemplation of the future, the formation of intentions, beliefs and purposes, or the exercise of choices. The question is simply whether the capacity for those functions was there at all. Diminished responsibility is not a defence known to New Zealand law.
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- I** And at p 254:

The common law does not recognize irresistible impulse as a defence. There is no requirement that the decision be free from irrationality, obsession, loss of

informal inhibitions or other mental abnormality predisposing the accused towards a particular course of conduct. As North P said in *Burr* at p 742, (quoting Richmond J in the Supreme Court): ‘a mind which ... was working well enough to ... put into effect the thought that had got into his mind (*however it had not there*) and to direct his body to actually put that design into actual practice and effect’ is sufficient (emphasis added). Compulsion in the sense used by Mr Gorringer is a common element to psychotic behaviour. An example is the man who kills his wife because he is driven to do so by voices in his head. Significantly, even in those cases the McNaughten rules are not addressed to the involuntariness of the conduct as such. Rather, the focus is upon the capacity to understand what he was doing or that it was wrong. The suggestion that the existing two limbs of the McNaughten rules should be joined by a third — lack of capacity to resist compulsive urges — has from time to time been argued but always rejected: *R v True* (1922) 16 Cr App R 164; *R v Flavell* (1926) 19 Cr App R 141; *R v Deighton* (1900) 18 NZLR 891. As was said in an explanatory note to the relevant cl 28(2)(b) in the New Zealand Crimes Bill 1989:

‘The clause does not attempt to deal with the vexed question of volition. It seems that there are people who know what they are doing, know that it is wrong, but are unable, because of psychiatric disorder, to stop doing it. How the law can provide for these cases without opening the floodgates to those who simply give in to temptation is a question that has so far defied a practical answer.’

Freedom of choice in the special sense that the mind is free from compulsive urges is not an essential element of intent. Its absence will not support the defence of automatism.

The defence of automatism ought not to be considered unless a proper foundation has been laid for it. In this regard, Lord Denning said in *Bratty v Attorney General for Northern Ireland* at pp 413–414:

My Lords, I think that the difficulty is to be resolved by remembering that, whilst the *ultimate* burden rests on the crown of proving every element essential in the crime, nevertheless in order to prove that the act was a voluntary act, the crown is entitled to rely on the *presumption* that every man has sufficient mental capacity to be responsible for his crimes: and that if the defence wish to displace that presumption they must give some evidence from which the contrary may reasonably be inferred. Thus a drunken man is presumed to have the capacity to form the specific intent necessary to constitute the crime, unless evidence is given from which it can reasonably be inferred that he was incapable of forming it, see the valuable judgment of the Court of Justiciary in *Kennedy v HM Advocate* 1944 SC (J) 171, 177 which was delivered by Lord Normand. So also it seems to me that a man’s act is presumed to be a voluntary act unless there is evidence from which it can reasonably be inferred that it was involuntary. To use the words of Devlin J, the defence of automatism ‘ought not to be considered at all until the defence has produced at least *prima facie* evidence’ see *Hill v Baxter* [1958] 1 QB 277, 285; and the words of North J in New Zealand ‘unless a proper foundation is laid,’ see *Reg v Cottle* [1958] NZLR 999, 1025. The necessity of laying the proper foundation is on the defence: and if it is not so laid, the defence of automatism need not be left to the jury, any more than the defence of drunkenness (*Kennedy v HM Advocate* 1944 SC (J) 171), provocation (*Rex v Gauthier* (1943) 29 Cr App R 113, CCA) or self-defence (*Reg v Lobell* [1957]

- A** 1 QB 547; [1957] 2 WLR 524; [1957] 1 All ER 734; 41 Cr App R 100, CCA) need be.
- What, then, is a proper foundation? The presumption of mental capacity of which I have spoken is a provisional presumption only. It does not put the legal burden on the defence in the same way as the presumption of sanity does. It leaves the legal burden on the prosecution, but nevertheless, until it is disproved, it enables the prosecution to discharge the ultimate burden of proving that the act was voluntary. Not because the presumption is evidence itself, but because it takes the place of evidence. In order to displace the presumption of mental capacity, the defence must give sufficient evidence from which it may reasonably be inferred that the act was involuntary. The evidence of the man himself will rarely be sufficient unless it is supported by medical evidence which points to the cause of the mental incapacity. It is not sufficient for a man to say 'I had a black-out': for 'black-out' as Stable J said in *Cooper v McKenna*, *Ex parte Cooper* [1960] Qd LR 406, 419 'is one of the first refuges of a guilty conscience and a popular excuse.' The words of Devlin J in *Hill v Baxter* [1958] 1 QB 277, 285 should be remembered: 'I do not doubt that there are genuine cases of automatism and the like, but I do not see how the layman can safely attempt without the help of some medical or scientific evidence to distinguish the genuine from the fraudulent'.
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- In *R v Cottle* [1958] NZLR 999, it was held that it is a question of law for the court to determine whether a proper foundation for the defence has been raised. A plea of automatism is not lightly accepted (see *R v Burr*).
- E** There are in law two types of automatism, namely, insane and non-insane automatism. The basis of the distinction lies in whether the cause is internal or external to the accused. *R v Hennessy* [1989] 2 All ER 9 and *R v Quick* [1973] QB 910 suggest that insane automatism arises where the primary cause of the abnormality is internal to the accused and prone to recur. The condition will be classified as a disease of the mind. It will then fall within the McNaghten Rules (see *R v Barry Douglas Burgess* (1991) 93 Cr App R 41) which is reflected in s 84 of the Penal Code. Non-insane automatism occurs where the abnormality is caused by a factor external to the accused like, for example, a blow to the head, medication, alcohol or drugs. The distinction affects only the burden of proof and not the matters that require to be proved or disproved. As I said in *Public Prosecutor v Kenneth Fook Mun Lee (No 1)* [2002] 2 MLJ 563 at pp 575–576:
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- As I said earlier, where the condition is a disease of the mind, it will fall within the McNaghten rules which is reflected in s 84 of the Code. This section, read with s 105, will place the onus on the accused to establish the defence. A similar view was taken in the case of epilepsy in *Sinmasamy v PP* [1956] MLJ 36. If the condition does not produce a disease of the mind, the onus will be upon the prosecution to exclude the alleged incapacity (see *Police v Bannin* [1991] 2 NZLR 237).
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- The alleged cause of automatism in this case is hypoglycaemia. It is not caused by a factor external to the accused. It will occur when there is a drop in the blood sugar of the accused and may recur. DW7 said that it could recur as the symptoms of hypoglycaemia can reappear after several years. Thus, I do not accept the evidence of DW9 when he said that a recurrence is unlikely as the attack that the accused had was the first and only one. The
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accused's condition therefore falls under the classification of insane automatism and the burden is on him to establish the defence on the balance of probabilities.

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Whether the accused was in a state of automatism

GL Glasgow in his article entitled *The Anatomy of Automatism* published in the New Zealand Medical Journal Vol 64, 1965 ('exh D97') in outlining the characteristic features of automatism said that to complete the picture it would be necessary to establish a cause for the state of automatism, such as a preceding epileptic attack, a head injury, hypoglycaemia or low blood sugar, or some other acceptable and identifiable cause of a transient disturbance of brain function. In this case, the accused was alleged to have been in a state of automatism at the material time. It is pertinent to first analyse the characteristic features of automatism, if any, exhibited by the accused at the material time before determining whether the accused was in fact undergoing a hypoglycaemic episode. This is appropriate as there must first be evidence to show that his act was involuntary before the cause of the mental incapacity can become relevant.

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I have outlined the facts of the case in considerable detail in an earlier part of the judgment. In order to resolve the issue currently under consideration, it is sufficient for me to identify the salient features of the facts that will illuminate the state of mind of the accused. On 22 August 2000 at about 1.45pm, the accused joined three of his friends at the Weng Seng Restaurant. They had lunch and some alcoholic drinks. The accused had five to six glasses of beer and was talkative. He left the restaurant in his car at about 7pm. He appeared normal and did not look drunk. He telephoned DW4 just after 7pm. He told her that he was on his way home and that he was alright. He told her not to worry. DW4 said that whenever the accused comes back home late from work he would telephone her. On the way back he would have paid toll at two points. Just before 8pm, his car was parked across the road at Jalan Istana Baru. He was standing on the middle of the road like a policeman manning a roadblock. He stopped a motorcyclist who sped off immediately. He then took out a gun from the ankle holster on his leg and fired two shots in the air. PW4, who was driving a taxi, was then stopped by the accused. In doing so, the accused pointed the gun at him. When PW4 stopped the accused went towards him. The accused asked PW4 to get out. When PW4 got out of the car the accused told him to stay there. The accused then pointed the gun in the direction of PW4's head and stomach. The accused told PW4 to stand straight. When he did so the accused signalled to him the 'Good' sign by showing the thumb. PW4 stood straight for about ten minutes. The accused then walked towards a Perdana car on the right of PW4. He looked normal, was steady and not staggering. He knocked at the windscreen and driver's side of the window of the Perdana with the gun asking the driver to come out. The driver did not come out. The accused tried to open the door and looked angry. He then moved to the front right side of the car and fired a shot which went through the driver's side of the Perdana. When PW6 shouted at the

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- A accused he fired a shot in the direction of PW6. PW6 asked everybody else to get out of their cars. The accused then fired shots at a military truck. He then went towards a parked taxi and was searching for the driver. The accused appeared confused and was walking aimlessly as if he was lost. He appeared to be unconscious of his surroundings. He pointed his gun at the crowd whenever they tried to approach him. The accused was steady before
- B his arrest. At the police station the accused slept on the floor for about 20 minutes and became incontinent. When PW15 saw the accused at about 10.05pm, he was aggressive and had a strong smell of alcohol. The accused kept referring to him as one Khoo Ah Si. The accused was taken to the hospital at 12.30am. When PW8 saw him he was agitated, restless, abusive and demanding to be freed of his handcuffs. He was alert and wanted to
- C contact his wife. The accused then wrote a note on a piece of paper (exh P17). He was not aware why the police had arrested him.

- I will now consider, against the background of the defence raised, whether the accused acted automatically in the sense that all the deliberative functions of his mind were absent at the time of the incident. In so doing,
- D I consider it necessary to refer to *R v Stone* (1999) Can Sup Ct Lexis 30 where it was held at p 46:

- I agree that the plausibility of a claim of automatism will be reduced if the accused had a motive to commit the crime in question or if the ‘trigger’ of the alleged automatism is also the victim. On the other hand, if the involuntary act is random and lacks motive, the plausibility of the claim of automatism will be increased. A question that trial judges should ask in assessing whether the defence has raised evidence which would permit a properly instructed jury to find that the accused acted involuntarily on a balance of probabilities is therefore whether or not the crime in question is explicable without reference to the alleged automatism. If this question can be answered in the negative,
- E the plausibility of the accused’s claim of involuntariness will be heightened. Such was the case in *Parks*, supra, for example, where there was no explanation for why the accused would attack his ‘in-laws’, with whom he otherwise had a good relationship, except automatism induced by a state of somnambulism. In contrast, if this question invokes a positive response, the plausibility of the claim of involuntariness will be decreased.

- G The facts of the case reveal that the accused fired two shots in the air when the motorcyclist sped away and at the deceased when she refused to come out of her car. He did not fire at PW4 as his instructions were obeyed. He fired at the deceased as she had disobeyed his instructions. If she had obeyed him, he would not have fired the shot. He pointed the gun at the
- H crowd only when they approached him. He had a telephone conversation with DW4 prior to the incident. This is something that he normally does when he returns home late as on that day. It is the conduct of the deceased that triggered the accused to shoot at her. He only fired at the deceased when his instructions were not obeyed. It was therefore not a random shot. This is supported by the fact that he did not fire at PW4 as his instructions
- I were obeyed. The sequence of events indicates that he had sufficient mental capacity to be responsive enough to make a decision. In my opinion, the expert medical evidence adduced by the defence does not militate against

this finding. When DW3, a leading authority on the field of hypoglycaemia, was asked whether the incident that happened was done with a conscious mind, he said:

I do not know what went on in his mind.

DW9, having said that the accused's state of automatism was preceded by a severe hypoglycaemia or low blood sugar attack which had caused a transient disturbance of his brain function making him unaware or unconscious of his acts which led to the fatal incident then said:

I maintain that once a person is in a hypoglycaemic state his consciousness is impaired. There are different levels of impaired consciousness. They are clouded consciousness, delirium, stupor and coma. In an altered state, there is no full awareness. In a clouded stage he may be partially aware.

This only shows that the consciousness of a person in a hypoglycaemic state is impaired. DW9's evidence is a medical exegesis of automatism with no reference to its legal connotations. DW7's evidence that the accused was unaware of what he was doing is no answer to the legal determination that has to be made on the mental state of the accused. In any event, their evidence is not supported by an analysis of the factual background. To that extent, DW3 is more forthright in his testimony. However, there can be no dispute that the accused's mind was in an impaired state when he fired the shot. But the authorities that I have referred to earlier reveal that the question that needs to be answered is whether the accused had retained sufficient mental capacity to reach the threshold required for the offence in respect of which he had been required to enter his defence. In view of the fact that diminished responsibility and irresistible impulse are not defences in Malaysia matters that go towards those defences cannot be taken into account. Thus, awareness of any degree will qualify however clouded, confused and distant that awareness might have been. It will be no defence that the accused's capacities might have been severely impaired that the normal inhibitions might be absent, that his decisions were not free from irrationality, or that there might have been a compulsive urge to act in a certain way. What is important is the existence of a sufficient degree of conscious volition in the accused in respect of what he did to make him legally responsible. The concept of mens rea has never been set so high as to require levels of perception and reasoning powers equalled with those of intelligent people or of persons only at their most acute (see *Haynen v Ministry of Transport* (1988) 3 CRNZ 587). As North P said in *R v Burr* at p 742:

And further, I tell you that it is no defence to him that his mind was lowered in efficiency; that he did not have his normal level of consciousness but was working at some lower level; that his mind was disordered in any of the ways suggested by the psychiatrists, because the law is merely concerned to see, as I have said, that he, being a man who was not actually asleep, had the purpose in his mind of killing Mr Faine and had a mind which though not as good perhaps as his normal mind, nevertheless was working well enough to design a rational plan to accomplish and put into effect the thought that had got into his mind (however it had got there) and to direct his body to actually put that

- A design into actual practice and effect. He will then have a sufficient degree of conscious volition in what he did to make him legally responsible.
- I would therefore hold that the accused was sufficiently conscious of what he was doing when he fired the fatal shot as a result of a decision made by him to do so following a refusal to obey his instructions. This means that
- B not all the deliberative functions of his mind were absent. The corollary is that the shot fired by the accused at the Perdana is explicable without reference to his alleged state of automatism. His mind, though impaired, was working well enough to put into effect the thought that had got into him. He had therefore fired at the deceased in a voluntary, though impaired, state of mind. Thus, the defence of automatism raised by the accused
- C cannot be sustained. The case of *Broome v Perkins* (1981) 85 Cr AR 321 shows that the fact that a person suffered from hypoglycaemia is not a relevant matter for consideration if at the material time he was conscious of what he was doing. Ordinarily, therefore, it would be unnecessary to go any further in view of my finding.
- D Be that as it may, it is necessary for me to determine whether the accused was indeed undergoing a hypoglycaemic episode at the relevant time in order to determine whether my finding of intoxication at the close of the prosecution case can be maintained. I have already ruled that automatism is not available as a defence to the accused. My ruling only means that he was aware of what he was doing. That does not mean that he
- E was not suffering from hypoglycaemia as a person undergoing an episode may still be aware of what he is doing. As DW3 said, a person who suffers from hypoglycaemia need not necessarily go into automatism. If it is found that there is no evidence to show that the accused was suffering from hypoglycaemia, it will explain the impaired mind of the accused and thereby facilitate a finding that he was intoxicated at the material time. Intoxication
- F was the alternative submission made by the defence. Thus, what is required is a consideration of the sufficiency of the evidence adduced by the defence to determine whether the accused did in fact suffer from hypoglycaemia. The defence relied on the evidence of DW2, DW3, DW6, DW7, DW8 and DW9 to establish hypoglycaemia. DW2 and DW6 are medical practitioners who had attended to the accused previously. DW3, DW7 and DW9 based
- G their evidence on the findings of DW2 and DW6 and the behaviour of the accused at the time of the incident. DW8 gave evidence on the state of intoxication of the accused at the time of the incident.
- DW2 saw the accused on four occasions. On the first two occasions, the blood reading of the accused was 3.9 millimoles per litre. DW2 said that he
- H made a provisional diagnosis of hypoglycaemia. He also told the accused that he ought to be investigated further for the cause of the hypoglycaemia and advised him to keep up his blood sugar by taking sugar frequently. On the third occasion when the accused saw DW2, he brought with him the results of blood tests that he had done. They did not disclose any hypoglycaemia. In my opinion the provisional diagnosis of hypoglycaemia
- I made by DW2 requires to be assessed carefully for several reasons. Firstly, DW2 did blood tests on the accused on 23 October 1991 and 30 October 1991. On both occasions he found the blood sugar level to be low. With

regard to the first test he said that the reading was 71mg which is 3.9 millimoles per litre. It was this reading on 23 October 1991 that made DW2 say:

There is a special significance in the reading of 3.9 millimoles on that day. It means that the patient has hypoglycaemia.

However, the calculation of DW2 is contradicted by the cross-examination of PW18 who said that 71mg is 3.94 millimoles per litre. No attempt was made by the defence to explain this contradiction. Perhaps DW2 had made a genuine mistake in making his calculation. But that does not preclude his evidence from being treated with extreme caution. The difference, though small, is significant in view of the evidence of DW3 and PW18 who had expressed doubts on a reading of 3.9 millimoles per litre itself as being indicative of low blood sugar. Secondly, PW18 said that the blood tests, being glucometer readings, should not be accepted for diagnosis purposes because of the variability of the result depending on the end user technique and the lack of quality control of the kit as opposed to laboratory readings and performance. The tests cannot therefore be the foundation for drawing any firm conclusion on their own. Thirdly, the diagnosis of hypoglycaemia by DW2 is only provisional. As DW3 said it is only suggestive of hypoglycaemia and he is surprised that no further investigations were done. As such, he is unable to confirm the diagnosis. DW7 said that the provisional diagnosis means that it must be proved before there can be a conclusive finding. PW18 said that it is inappropriate for a diagnosis to be made without supporting evidence. She also said that if DW2 had found the blood reading of the accused to be significant any physician of his standing would have referred the accused for further assessment which was not done. Fourthly, the blood results produced by the accused on his third visit to DW2 clearly showed that his low blood sugar was no longer a problem. This ought to have alerted DW2 on the provisional finding of hypoglycaemia that he had made earlier. But it did not. Fifthly, DW2 ought to have followed up his provisional finding with the necessary tests to identify the cause of the low blood sugar. A more prudent course to have been adopted would have been to identify the cause of the hypoglycaemia. DW2 realized this but did not do anything about it. Instead he advised the accused to take sugar frequently to keep up his blood sugar. This advice would be extremely dangerous if the cause of the low blood sugar was diabetes. DW2, a senior physician, would have been aware of this. The accused ought to have been advised to take sugar only if the blood sugar level falls and not to keep it up. Sixthly, the evidence of the finding of hypoglycaemia by DW2 as being provisional is contradicted by the testimony of the accused who said that DW2 had 'confirmed that I was definitely suffering from low sugar in the blood' and DW4 who said that 'Dr Ridzwan said that it is confirmed that Kenny has low blood sugar' This contradiction was not explained. On the other hand, DW4, in an attempt to strengthen the low blood sugar condition of the accused, further weakened it when she described what she and the accused had for tea even before their first visit to DW2. As she said,

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- A** We would normally have tea at about 5.00 pm. The tea will be with condensed milk and glucose and anything sweet. On that day I cannot remember what kind of tea we had. But definitely we had tea.

- Even though, on being questioned by the court on this part of her evidence, DW4 said that she had made a mistake and that the accused will have tea without glucose she made no reference to her evidence on the consumption of condensed milk by the accused. Clearly the evidence of DW4 is a misguided embellishment to add a sweet flavour to the complaint of low blood sugar of the accused. In the circumstances I am not inspired to place much weight or significance on the provisional finding of low blood sugar of the accused by DW2.

- C** The evidence of DW6 requires careful scrutiny. He said that the accused first saw him on 1 October 1991. Just prior to this visit DW4 rang up DW6 to say that the accused was having a faintish-like feeling. DW6 advised DW4 to give the accused a sweet drink. She did so and the accused recovered. Soon after that they went to DW6's clinic. Without doing any tests DW6 diagnosed the accused as suffering from simple heartburn and low blood sugar. The evidence of DW6 must mean that DW4 knew DW6 and that she had gone to his clinic. In this regard DW4, in her re-examination, said:

- E** I heard of Dr Thiagi. He is one of my husband's doctors. I am not aware of my husband's visits to Dr Thiagi.

DW4 was recalled by the defence at the close of its case to explain her evidence. She was only asked to clarify the part where she had said that she was not aware of her husband's visits to DW6. As she said:

- F** The question that was posed to me the last time was whether I was aware of all the visits to Dr Thiagi. My answer was I was not aware of all the visits. I am aware of some visits of my husband to Dr Thiagi.

- She then described the events leading to her visit to DW6's clinic with the accused on 1 October 1991. The clarification offered by DW4 does not explain away her previous testimony. If her answer on the previous occasion was that she was only '... not aware of all the visits' of the accused to DW6's clinic it follows that she must still have known DW6. In that event there was no reason for her to have said on that occasion that she '... had heard of Dr Thiagi.' If she had taken the accused to DW6's clinic she would have known DW6 and not just heard of him. As this part of her evidence was not explained when recalled I am unable to accept her explanation with the result that the evidence she gave on the previous occasion remains. In view of the glaring inconsistency between the evidence of DW4 and DW6 on this visit, I am unable to accept DW6's evidence that the accused visited him on 1 October 1991 with the specific complaint as alleged. This finding explains why no entry was made on the diagnosis made by DW6 on that day on exh D91 though he had recorded the complaints. It is indeed strange that DW6 made no record of his diagnosis when he himself admitted in cross-examination that any examination done must be recorded. A close perusal of exh D91 further reveals that the entry made on exh D91 with regard to

the complaint of syncope-like feeling for the date 1 October 1991 is with a different pen from the main entry for some other complaint and has been inserted on available empty spaces. In the circumstances it is my firm finding of fact that the evidence given by DW4 when she first testified is the correct one and, consequently, the accused did not visit DW6 as claimed. I am therefore driven to conclude that the evidence of DW6 is an after-thought. DW6 also gave evidence on the accused's visit to him on 12 November 1998. The entry for this visit reads, inter alia, as follows:

12 November 1998 — Hypoglycaemia — syncope — Adv to see Ridzwan.

I find this entry suspicious in view of inconsistent answers given by DW6 in relation to it. At one stage he said that when he wrote hypoglycaemia leading to syncope he was referring to the finding made by DW2. That means that the information was supplied to DW6 on the very day he made the entry. But at another stage of his evidence DW6 said that it was two days later that the accused told him that DW2 had confirmed that his blood glucose was low. Yet at another stage he said that he did not know that DW2 had made a finding of provisional low blood sugar. Furthermore, the entry is below the last line where one normally does not write. Again I am compelled to conclude that this part of the evidence of DW6 is an after-thought. In the circumstances I reject the evidence of DW6 on the low blood sugar condition of the accused. As a matter of fact, the accused himself admitted to PW8 in the hospital that he had no medical illness previously. This admission was not explained by him. Though he said that he cannot remember the conversation with PW8 he did not go further to say that what he is claimed to have admitted is untrue or a mistake. An admission which is not explained must be accepted (see *MA Clyde v Wong Ah Mei & Anor* [1970] 2 MLJ 183). This is provided by s 31 of the Evidence Act 1950. In commenting on the section *Field's Law of Evidence* (12th Ed) Vol 2 says at p 1914:

The provision that admissions may operate as estoppels is sometimes attempted to be used as if, instead of saying that an admission is not conclusive proof, the section said that an admission is not sufficient proof without corroboration. But this is not the meaning. All that s 31 provides is that an admission, unless it operates as an estoppel is not conclusive. The person against whom it is proved is at liberty to show that it was mistaken or untrue. It is true that evidentiary admissions are not conclusive proof of the facts admitted and may be explained or shown to be wrong, but they do raise an estoppel and shift the burden of proof on to the person making them or his representative-in-interest. Unless shown or explained to be wrong, they are an efficacious proof of the facts admitted (*Avadh Kishore Dass v Ram Gopal* AIR 1979 SC 861).

I do realize, however, that an admission made by a party when drunk is not of such weight as one made when sober but it is still admissible (see *R v Hedges* 3 Cr App R 262; *R v Saw Min* AIR 1939 R 219). I fail to understand why the accused, who claimed to be aware of his hypoglycaemic condition from 1991 and in respect of which he had even kept glucose in his office and which caused him to have a blackout in the Awana incident did not mention

A this to PW8. His admission to PW8 must therefore be true with the result that he had admitted that he had no hypoglycaemia. My findings on the evidence of DW2 and DW6 coupled with the admission made by the accused mean that there is no evidence to show that the accused had a history of hypoglycaemia.

B In view of my findings on the evidence of DW2 and DW6 the opinions expressed by DW3, DW7 and DW9 must be assessed against that background. It is pertinent to observe that a large part of the evidence of DW3 and DW7 is based on the evidence of DW2 and DW6 while the evidence of DW9 is based, in addition, on other factors including interviews he had with the accused. Such evidence is not of much value compared with the evidence of doctors who had attended to the patient (see *Gopessur v Bissessur* 16 CWN 265; *Monosseh v Shapurji* 10 Bom LR 1004). As *Sarkar on Evidence* 15th Ed says at p 901,

D Opinion of medical men based on hearing the evidence of other witnesses without observing facts is not of much value. Thus opinion of doctors based mostly on prescription and on their general knowledge of the course of a disease are always indirect evidence and not of much weight when compared with the evidence of doctors who personally attended the patient (*Hari Singh v Lachmi* 59 IC 220; 12 PLR 1921).

The significance of the evidence of DW2 and DW6 to the opinion expressed by the experts can be comprehended with ease. The experts were unanimous in the view that the behaviour of the accused at the time of the incident can be similar to one under intoxication. Thus, as PW18 said, in order to distinguish between intoxication and hypoglycaemia one has to fall back on the biochemical evidence because of the overlap of the symptoms. DW7 said that in the absence of blood sugar measurements it would be very difficult to differentiate between hypoglycaemia and intoxication. Similarly PW19 said that in forensic psychiatry a final diagnosis must be based on a mental state examination and laboratory findings. The previous medical history of the accused is therefore of critical importance to rule out intoxication. Accordingly, the experts relied on the evidence of DW2 and DW6 to identify the cause of the accused's behaviour. In this regard DW3 said,

G His medical history is certainly suggestive of episodic hypoglycaemic attacks of undetermined origin.

DW7 said,

H He attended the Pantai Medical Centre. His blood sugar was low. His symptoms were relieved by taking glucose. The next morning he attended Dr Ridzwan's clinic complaining of chest pains, heart palpitation, numbness of the hands and sweating. His wife testified that he had breakfast at 7.00 am. To me these symptoms in 1991 are suggestive of symptoms related to the autonomic nervous system or the neurogenic symptom.

I She also said that the accused's history of provisional findings mean that he is not a healthy person and is thereby suggestive of hypoglycaemia. DW9, in saying that the accused had a convulsion in the police station, said,

I take that to be evidence of convulsion in view of his history.

The legal position of evidence upon which an expert bases his opinion is well settled. Where an expert relies on the existence or non-existence of some fact which is basic to the question on which he is asked to express his opinion, that fact must be proved by admissible evidence (see *English Exporters (London) Ltd v Eldonwall Ltd* (1973) 1 All ER 726; *R v Abadom* (1983) 1 All ER 364). As Miles CJ said in *Forrester v Harris Farm Pty Ltd* (1996) 129 FLR 431 at p 438,

It is a trite principle of evidence law that the opinion of an expert, whatever the field of expertise, is worthless unless founded upon a substratum of facts, which facts are proved by the evidence in the case, exclusive of the evidence of the expert, to the satisfaction of the court according to the appropriate standard of proof. Whether or not the expert believes in the substratum of facts or knows them to be true or is satisfied that they are true, is completely beside the point. The expert's function is to express an opinion based on assumed facts, not to express a view on whether the assumptions are justified.

The grounds upon which an expert opinion is based may therefore be properly be inquired into (see *Pacific Tin Consolidated Corporation v Hoon Wee Thim* (1967) 2 MLJ 35). In *Brij Basi v Moti Ram* AIR 1982 All 323 it was held that if the oral evidence leads to a positive conclusion one way or the other the opinion of experts have to yield or have to be accepted or rejected in accordance with the finding arrived at on an appraisal of the direct oral evidence. If such direct oral evidence is not accepted the opinion expressed by the expert will collapse. In this regard I refer to *Sarkar on Evidence* 15th Ed where it says at p 878,

Opinions or inferences must be based on certain premises. These premises may come to the witness' knowledge from personal observation, eg when a medical man has examined the body and performed autopsy, or, they may be supplied to him by the testimony of others. Here the premises must be stated hypothetically and accurately in order to obtain his opinion on the hypothetical data. Hypothetical questions should generally be based on facts proved in the case by testimony. Hypothetical questions may also be based on facts assumed for the time being to be true, but the premises must afterwards be supplied by the testimony of others. The danger of such assumption is that the jury or the witness might be misled and take as proved what is merely an assertion on the part of the lawyer. If the premises are afterwards found to be untrue, the opinion of the witness must be rejected.

My rejection of the evidence of DW2 and DW6 therefore means that the foundation of the evidence of DW3, DW7 and DW9 has been destroyed insofar as it is based on the evidence of DW2 and DW6 thereby rendering that part of their evidence worthless. Be that as it may, in the absence of the evidence of DW2 and DW6 the best evidence that the accused could have adduced in proof of his defence is the 72-hour fasting test. The effect of this test was explained by DW7. It is difficult to establish hypoglycaemia at a prior point of time without proper medical tests. In support I refer to exh D81 where it says at p 104,

From the legal point of view, it is difficult if not impossible to establish, in retrospect, whether a subject was hypoglycaemic at the relevant time especially if they were not receiving insulin or sulphonylurea therapy at the

A time. In the cases that have come to court and been reported in the medical literature the defence rested largely upon the clinical history and demonstration or reactive hypoglycaemia during an oral glucose load test.

However, the evidence of DW3 and DW9 show that the accused did not undergo any tests on the ground that he could not avail himself of one. This explanation cannot be accepted as the evidence of DW10 makes it clear that a prisoner may obtain private medical opinion. In any event the accused could have sought a court order for that purpose if he was so minded but he did not do so. I must add that this does not mean that I am drawing an adverse inference against the accused. That can be done only for withholding or suppressing evidence and not for a failure to obtain evidence as in this case (see *Munusamy v Public Prosecutor* [1987] 1 MLJ 492). But the fact remains that in failing to do so the accused has deprived himself of the best evidence that may have established his case. Thus DW3 and DW7 said, even with a consideration of the evidence of DW2 and DW6, their opinion is only a possibility in the absence of further tests having been done on the accused. I pause to add that it is superfluous to state that in a civil case we are concerned with probabilities, not with possibilities (see *TNT Management Pty Ltd v Brooks* 23 ALR 345). As the burden of proof to establish hypoglycaemia on a balance of probabilities is on the accused the evidence led must meet the required standard. Therefore the evidence of DW3 and DW7 where they say that their conclusion is only a possibility is of no avail. DW9's evidence that he is 100% sure that the accused had hypoglycaemia at the material time cannot also be accepted in the absence of any tests having been conducted on the accused as explained by DW3 and his own earlier evidence that nobody in this world can be 100% certain of anything.

I shall now consider certain other evidence of significance relied on by the experts to arrive at their finding. DW3 referred to the waking up habit of the accused and said that it is a strong pointer towards an organic cause of hypoglycaemia. In this regard DW4 had testified that the accused had this problem for the last ten years. I find it difficult to associate this habit with hypoglycaemia as opined by DW3 as the recorded blood sugar reading of the accused was low only in 1991. The readings thereafter were normal. When the readings were normal the waking up habit must have also returned to normal as hypoglycaemia would not have been a problem then. But DW4's evidence shows that the habit continued. That, to my mind, is illogical. The habit is perhaps explained by the fact that the accused, as testified by DW4, goes to sleep normally between 1–2am. Surely a person who goes to sleep late will find it difficult to get up early in the morning. DW9 also referred to the finding of PW13 that the accused had gunpowder residue on his left web. He said that this indicates that the accused had used his left hand to shoot which is not consistent with alcohol as the accused is right handed. However, PW13 also said that one has to be careful in interpreting the deposition of the gunshot residue on a specific hand to link it with the hand that was used to shoot as his activities after the shooting incident may result in the loss or subsequent re-distribution of the gunshot residue originally deposited. In the absence of evidence to show that there

was no re-distribution of the gunshot residue it is difficult to accept that the accused used his left hand to shoot. On the contrary the subsequent conduct of the accused may indicate that there was such a re-distribution. DW9 also said that the snoring of the accused at the police station meant that he had a quiet convulsion. PW19 disagreed with this view as snoring can be caused by other conditions like sleep apnea. It cannot therefore be accepted as an indicator of hypoglycaemia. The time taken by the accused to recover at the police station till he saw PW8 at the hospital was slow. DW3 said that in cases of hypoglycaemia the recovery is remarkably quick while in the case of intoxication it is very slow. DW9 said that after recovery from hypoglycaemia a person will be normal. PW18 said that if the behaviour of the accused was due to hypoglycaemia the recovery he had would have been full. Thus, she said, the behaviour of the accused after he recovered in the police station is not consistent with hypoglycaemia. She added that if a person had been severely hypoglycaemic leading to unconsciousness he would not wake up without assistance. The slow recovery of the accused thus supports the view that the accused was not hypoglycaemic. The result of DW9's interviews with the accused is also insignificant in the absence of any positive supporting medical evidence. Similarly the evidence of DW3, DW7 and DW9 that the seven characteristics as specified in exh D97 have been satisfied to suggest hypoglycaemia is not complete without evidence of the cause of the state of automatism as stated in that exhibit itself.

That leaves for consideration the evidence of DW8 which may suggest that the conduct of the accused at the time of the incident was not due to intoxication. He calculated the blood alcohol concentration in the accused at about 8pm to be in the range of about 92–119mg based on the blood and urine alcohol concentration from samples taken at 1.20am. The blood alcohol concentration was 198 mg per 100ml of blood and the urine alcohol concentration was 25 mg for 100ml of urine. DW8 said that as the urine alcohol level is higher than the blood alcohol level that was the peak period of intoxication of the accused. At any point of time prior to that the blood alcohol level will be on the ascending scale. One of the factors that DW8 considered in making his calculation is the time difference between 8pm and the time the samples were taken, that is to say, at 1.20am. That, to my mind, raises a significant problem. DW8 said that his calculation showed that the equilibrium peak was reached around the time the blood and urine samples were taken. He had earlier said that the peak was between 12.00 and 1.00 am. With such evidence I fail to understand why the time of 1.20 am was used by DW8 to make his calculation. He did not explain why the earliest point of time at which the peak was reached was not used for the purpose of his calculation. This creates a doubt in the calculation made by DW8. He also said that the manner of calculation of the blood alcohol label is dependent on certain variables such as the amount of food in the stomach and the amount of fluid in the body. He said that a strong alcoholic drink will increase the fluid content in the body. As to whether six glasses of beer can produce this situation he said that it depends on the spacing of the drinks. The evidence adduced in this case shows that the six glasses of beer

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- A consumed by the accused was well spaced out. The absorption of alcohol in the accused must therefore be faster. When DW8 was referred to the food consumed by the accused on that day DW8 said,

If all that food is in the stomach the absorption will be even faster.

- B Again the absorption of alcohol in the accused must be faster. DW8 was referred by the prosecution to a book entitled *Drink, Drugs & Driving* where it is stated at p 17 that the additional effect of food is relatively much less important in the case of beer. DW8 disagreed with this view. This is in direct contradiction to his earlier evidence when he said that it is a strong alcoholic drink that will increase the fluid content in the body. Beer is not a strong alcoholic drink. It follows that the opinion expressed in the book referred to DW8 is more acceptable. In view of the variables involved in calculating the blood alcohol level at a prior point of time DW8 himself agreed that it is dangerous and not wise to do so. PW13 had also expressed a similar view. DW8, having recognized this problem, did not go further to specify the precise variables that must be considered and the exact variables that he took into account. This renders his evidence weak. Finally, the Widmark Formula which DW8 used to determine the amount of beer consumed by the accused is based on assumptions as stated in an article by him which is in [1995] 3 MLJ xli. It can therefore be open to errors. In this case the error is glaring by virtue of the fact that while DW8's calculation showed that the accused had consumed six to seven glasses of beer the oral evidence reveals that it was only six glasses. On the facts of this case, therefore, the evidence of DW8 must be viewed with caution. In any event the blood alcohol level of the accused at 8pm based on the calculation made by DW8 cannot be a positive indication of his state of intoxication. DW8 had himself conceded that different people react differently in order to be affected by alcohol. In the circumstances I am unable to accept his evidence. Thus the evidence of DW8 is of no assistance to show that the accused was not affected by alcohol at the material time.

The result is that the accused has failed to establish that he was undergoing a hypoglycaemic episode at the material time. This finding does not mean that the testimony of the defence medical experts is suspicious. They proceeded on the basis of an acceptance of the evidence of DW2 and DW6 which they are entitled to do and even opined that the accused ought to have been subjected to further examination. On the other hand I had analysed the evidence of DW2 and DW6 on a legal basis which I am required to do. It is just that the experts' views cannot be accepted as a result of the demolition of the foundation of their testimony.

- H In the light of my finding on hypoglycaemia the alternative defence submission of intoxication becomes a live issue. In view of the similarity of the symptoms of hypoglycaemia and intoxication as testified by the experts the behaviour of the accused at the material time must have been caused by intoxication. I had called upon the accused to enter his defence on that basis. With my rejection of hypoglycaemia the reason for the accused's slow recovery and incontinence at the police station becomes clear. DW3 said that the slow recovery and incontinence of the accused is consistent with

intoxication. The accused's incontinence will throw light on the stage of his intoxication at the time of the incident. In accordance with the Dubowski Chart it would have been at the upper level. This means that his blood alcohol level at the time of the incident would have been very much higher than what was revealed by the samples taken at 1.20 am. It follows that he would have had more than six glasses of beer. In any event, as I said in an earlier part of the judgment, the defence of intoxication can be established without the support of scientific evidence. Thus having considered the whole of the evidence adduced it is my firm finding of fact that the accused was in a state of intoxication at the time of the incident. I am therefore satisfied, on the balance of probabilities, that the state of intoxication of the accused at the material time, which I have explained in an earlier part of the judgment, precluded him from forming the necessary intention.

However, in finding that a case had been made out against the accused under cl (d) I had proceeded on the basis that it is the direct consequence of intention having been negated by intoxication that rendered the other parts of s 300 inapplicable. My research has now raised the question of the manner of ascertaining the existence of intention where intoxication is in issue, that is to say, whether such a determination must first be made without taking intoxication into consideration. I had brought this view to the attention of both parties in their submissions at the end of the defence case. They agreed with what I have set out below as the correct approach to determine intention in a case involving intoxication.

It is settled law that an inference of intention can be rebutted by evidence of intoxication pursuant to s 86(2). In cases where intoxication is in issue, the court must determine whether the accused had the requisite specific intent in the light of his intoxication (see *R v Sheehan* (1975) 2 All ER 960); *Kennedy v HM Advocate* (1944) SC (J) 171; *Bratty v Attorney General for Northern Ireland*). The critical matter for deliberation is whether a finding on the relevant mens rea must first be made before taking intoxication into consideration. This will have a determinative effect on the precise limb of s 300 that is applicable in cases of this nature. The onus of proof where intoxication is raised as a defence is on the accused. This is provided by s 105 of the Evidence Act 1950 which deals with the question of burden of proof where, inter alia, intoxication is a defence. The section reads as follows:

When a person is accused of any offence, the burden of proving the existence of circumstances bringing the case within any of the general exceptions in the Penal Code, or within any special exception or proviso contained in any other part of the same Code, or in any law defining the offence, is upon him, and the court shall presume the absence of those circumstances.

In commenting on the words 'the court shall presume the absence of such circumstances' in the section *Sarkar on Evidence* 15th Ed Vol II says at p 1529,

Section 105, in using the words 'shall presume the absence of such circumstances' requires the court to regard such absence as proved unless and until it is disproved or in other words their presence is proved (see s 4). S 105

- A** constitutes a departure from the ordinary rule that the onus of proving the guilt lies on the prosecution and makes it obligatory upon the courts to presume the absence of such circumstances as may bring the accused within some general or special legal exception, unless, and until their existence is shown by the accused or is admitted by the prosecution itself.
- B** The court is therefore bound, in the first instance, to presume the absence of such circumstances (see *Nga Tun Baw v Emperor* (1912) XVII IC 800). Thus, for example, the court has to regard the absence of grave and sudden provocation until the contrary is proved by the accused on whom the onus lies (see *R v Upendra* 19 CWN 653; *Pasadi Verabbayi v State of Andhra Pradesh* 1984 Cri LJ 440). Where therefore s 86(2) is in issue the court shall
- C** presume the absence of intoxication unless the defence adduces evidence to the contrary. In *Nga Tun Baw v Emperor*, it was held that the intention of a person can be explained by his state of intoxication. This accords with the common law position which was succinctly explained by Lord Denning in *Bratty v Attorney General for Northern Ireland* at p 413:
- D** My Lords, I think that the difficulty is to be resolved by remembering that, whilst the ultimate burden rests on the crown of proving every element essential in the crime, nevertheless in order to prove that the act was a voluntary act, the crown is entitled to rely on the presumption that every man has sufficient mental capacity to be responsible for his crimes; and that if the defence wish to displace that presumption they must give some evidence from which the contrary may reasonably be inferred. Thus a drunken man is
- E** presumed to have the capacity to form the specific intent necessary to constitute the crime, unless evidence is given from which it can reasonably be inferred that he was incapable of forming it
- This burden on the accused may be discharged by evidence of witnesses for the prosecution as well as by witnesses for the defence (see *In re Kali Charan Mukharji* 11 Cal LR 232). It follows that the court must first make a finding
- F** on the relevant mens rea before embarking on a consideration of the defence of intoxication.
- I shall now consider the mens rea under which the accused acted. Before doing so it is necessary to consider the distinction between intention
- G** and knowledge. Generally stated intention is the purpose or design with which an act is done. It is the fore-knowledge of the act, coupled with the desire of it. Knowledge is an awareness of the consequences of the act. There is a close relationship between intention and knowledge. As Chandrasekhara Aiyar J said in *Basdev v State of Pepsu* AIR 1956 SC 488 at p 490:
- H** In many cases intention and knowledge merge into each other and mean the same thing more or less and intention can be presumed from knowledge.
- In *R v Gora Chand Gopee* 5 WR 45, Peacock CJ in dealing with cl (d) of s 300 of the Indian Penal Code said at p 46:
- I** From the fact of a man's doing an act with the knowledge that he is likely to cause death, it may be presumed that he did it with the intention of causing death, if all the circumstances of the case justify such a presumption.

In *Rama v State* AIR 1969 Goa 116, Jetley J in commenting on the circumstances in which a consequence is deemed to be intended said at p 122:

It may be added that intention also includes foresight of certainty. A consequence is deemed to be intended though it is not desired when it is foreseen as substantially certain.

In *R v Nedrick* [1986] 3 All ER 1, it was held that a court may infer that a result is intended, though it is not desired when :

- (a) the result is a virtually certain consequence of the act, and
- (b) the accused knows that it is a virtually certain consequence.

In further amplification of this ruling, reference may be made to *Sarkar on Evidence* (15th Ed) Vol 1 p 326:

It is a universal principle that when a man is charged with doing a wrongful act, without any legal justification, of which the probable consequence may be highly injurious, the intention is an inference of law resulting from the doing of the act.

Thus, knowledge of a consequence which is virtually certain will be evidence of intention. An intention can be inferred from the nature of the act of a person (see *R v Monkhouse* (1849) 4 Cox CC 55). The nature of the weapon used and the part of the body to which the injury is caused may also facilitate the drawing of such an inference. In this case the accused fired the shot from outside the car into its driver's side through the front windscreen. The shot hit the upper part of the body of the driver where the vital organs of the body are situated. The shot is virtually certain to cause death and the accused must be presumed to know this. The act of the accused in firing the shot was deliberate as it was done following the refusal of the deceased to open the door of the car. The deliberate use of a dangerous weapon at another leads to the irresistible inference that it was done with the intention to cause death (see *Tham Kai Yaw & Ors v Public Prosecutor* [1977] 1 MLJ 174). Furthermore, the act of the accused in firing the shot was not one of accident or negligence. In *Behari v State* AIR 1953 All 203, it was held that where the injury caused is not the result of accident or negligence a strong presumption arises that the injury caused was intended to be caused though this presumption may be rebutted. That would be so even if there was no intention to cause death. In this regard, I refer to Gour's *Penal Law of India* (11th Ed) which says at p 2436:

For the commission of the offence of murder it is not necessary that the accused should have the intention to cause death. It is now well settled that if it is proved that the accused had the intention to inflict the injuries actually suffered by the victim and such injuries are found to be sufficient in the ordinary course of nature to cause death, the ingredients of clause *Thirdly* of sec 300 of the Indian Penal Code are fulfilled and the accused must be held guilty of murder punishable under s 302 of the Code.

The circumstances in which the shot was fired therefore go beyond mere knowledge and it must be inferred or presumed that it was done so

- A intentionally. Thus, the facts of the case fall within s 300(c) where intention is in issue. Clause (d) only applies where no other part of s 300 is applicable. Thus in a case like this where the intention of the accused as inferred from the result of his act could at least be that of causing such bodily injuries as the accused would have known to be likely to cause death cl (d) will have no application. As Addison and Din Mohammad JJ said in *Hasta Ismail v Emperor* AIR 1937 Lah 593 at p 595:
- B

Another matter on which we consider the Sessions Judge has gone wrong is the use of Cl 4thly, S 300, while convicting the accused. It has often been pointed out by this court that this clause comes into play only if no other clause applies and in a case like this where the intention of the assailants as inferred from the result of their act could at least be that of causing such bodily injuries as the assailants would have known to be likely to cause the death of their victim, the invocation of Cl 4thly was altogether erroneous.

C

In *Behari v State* AIR 1953 All 203, it was held that cl (d) does not apply to cases where a person intends to inflict an injury likely to cause death. The charge against the accused under cl (d) cannot therefore stand as the applicable section is s 300(c).

D

The presumption of the absence of intoxication which facilitated a finding that the accused acted with intention as envisaged by s 300(c) is displaced by the evidence of intoxication on record to show that he did not have the requisite intention. This means that the act of the accused which gave rise to an inference of intention has been rebutted. However, the fact remains that it is a case which initially fell under s 300(c). It is on that basis that s 86(2) became applicable. It is also on that basis that cl (d) became inapplicable. However, the facts are sufficient to make out a case under s 304(b) which does not require an intention to cause death or to cause such bodily injury as is likely to cause death. Such a course was adopted in cases such as *Suba Singh v Public Prosecutor*; *Tan Hung Song v R* and *Public Prosecutor v Teo Heng Chye*. Thus, I am unable to agree with the submission of the prosecution that the facts of the case disclose a case under cl (d). Accordingly, I find the accused guilty of an offence under s 304(b).

E

F

In his mitigation, learned counsel for the defence contended that the accused has no previous convictions and that he is very remorseful over the incident. The accused had co-operated with the police. He was a racing driver and had taught defence and advanced driving to the police and customs. He has made donations to organizations like Pemadam and has participated in numerous road safety campaigns. Learned counsel added that the impaired mind of the accused at the time of the incident must be considered. He referred to some authorities and said that like cases must be treated alike. The learned deputy public prosecutor urged the court to impose the maximum sentence in the interests of the public in view of the seriousness of the offence.

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The offence for which the accused has been found guilty is very serious. It is aggravated by his self-induced state of intoxication. Shooting at a person in that condition is certainly a dangerous act and a deterrent sentence must be imposed. Hence, the plea in mitigation advanced on

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behalf of the accused has to give way to the public interest to be served. Bearing in mind the sentence imposed in cases such as *Suba Singh v Public Prosecutor* and *Tan Hung Song v R*, I convicted the accused and sentenced him to eight years imprisonment to take effect from the date of his arrest.

Accused convicted and sentenced to eight years' imprisonment with effect from the date of arrest.

Reported by Mariette Peters-Goh

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